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BLOOD GROUPS OF NORTH QUEENSLAND ABORIGINES, WITH A STATISTICAL COLLECTION OF SOME PUBLISHED FIGURES FOR VARIOUS RACES.

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AT the suggestion of Dr. Heydon I undertook the examination of a number of full blooded aborigines at the Palm Island Settlement. Herein are published the results of this examination, a contribution to the pioneer work of Tebbutt and McConnel[8] [9] and also a collection of as many results obtained in various parts of the world as could be found in the literature available.

Technique.

The Palm Island Settlement lies about forty miles north-north-east from Townsville, which fact together with the irregularity of the steamer service increased somewhat the complexity of the technique. The open-slide method of Vincent[1] as modified by Heydon and Murphy[17] for work in the tropics was employed. The blood samples were collected by thumb puncture into half inch test tubes four-fifths full of 3·8% sodium citrate solution and allowed to settle for a period of about four hours. If the sample was a poor one, centrifuging by hand was employed. The supernatant fluid was then decanted, the corpuscles resuspended in 0·85% saline solution (test tube four-fifths full) by inversion and the mixture again allowed to settle for about three hours, when the supernatant fluid was decanted. A drop of the sediment was mixed with a drop of each of the test sera (Group II. and Group III.) at opposite ends of the slide, the slide placed on two tooth picks or matches on a moist filter paper and covered with half a Petri dish. The drop was well mixed by movements of the slide after five minutes and again after another five. The result was then read.

Sera for testing were taken from known Group II. and Group III. people. For a number of cases my own (Group II.) serum was employed, because

the regularity of its reaction was known, and the risk thereby obviated of selecting a donor whose serum did not possess the agglutinin β to the full or any degree.[15] [16] This precaution is advisable in all routine examinations. When different donors were used whose corpuscle reactions alone were known, the sera were often controlled by employing corpuscles of known reaction, though this was not always carried out. When such untried donors are used, it is advisable to collect blood from more than one donor of each group in case one should prove irregular.

Diagnosis.

Typical reactions are readily recognized with the unaided eye and in such cases the microscope was not used as part of the routine, though any doubtful appearances were always examined microscopically. The moist chamber method employed removes the difficulty experienced by Bais and Verhoef.[14] Many of these appearances were found to be due to rouleau formation, but this was easily detectable. More puzzling is an appearance noted quite well in one instance and possibly though doubtfully in more. This consisted of a number of small clumps of corpuscles, half a dozen or so in each, revealed by the microscope in a drop which appeared macroscopically to be free from agglutination. Of ninety specimens that yielded no reaction, and which were examined microscopically, only one gave a definite appearance of this nature.

Lattes and Cavazzuti[15] give as the criteria for "pseudo-agglutination" as distinguished from true agglutination: lack of specificity, absence of fixation of the agglutinin, inhibition by lecithin and low titre. The macroscopic appearances are various: "Commonly they were mediocre and slow; in some were seen, in addition, among the irregular masses, distinct rouleau formations. But it must be admitted that in certain cases intense and rapid agglomerations were seen, perfectly analogous in their appearance to a strong and authentic isoagglutination."[15] They offer no explanation of the phenomena, biological or otherwise (". . . *la vraie nature ne peut être mise en évidence que par des recherches soigneuses et complexes*"), so that the position of these reactions, defined only by negative characters is somewhat vague. Is this appearance I am describing caused by pseudo-agglutination or can it be explained by supposing that the corpuscles contained the agglutinogen *A* in very small amounts or were only slightly susceptible to the action of the agglutinin α?

The classification of the blood of one other subject was somewhat obscure. This was a girl of five whose corpuscles agglutinated strongly with Group II. serum, weakly with Group III. serum. Retested against two other samples of Group II. serum and one other of Group III. serum they agglutinated well with the former and not at all with the latter (the diagnosis was confirmed by the microscope). May this phenomenon be explained according to the findings of Lattes and Cavazzuti, by supposing that the corpuscles contained in very weak amount the agglutinogen *A*, so weak that it could be detected only by a serum with a high concentration of α agglutinin (the second Group III. serum proved to be typical when tested with other corpuscles whose grouping was known)?

It may be well worth while introducing the use of lecithin into routine examinations, if its differential action on pseudo- and true agglutination is what Lattes[15] claims for it.

I should like to record also, whilst speaking of the work of Lattes and Cavazzuti, that four out of the six samples of Group IV. corpuscles found in this series of examinations induced noticeably less agglutination with Group II. serum than with Group III. serum, the agglutination with Group II. serum being less also than that of Group III. corpuscles with the same Group II. serum. This is a further indication of a variation in titre of agglutinogens in the corpuscles.

Stability of Agglutinogens and Agglutinins.

In the examination of the aborigines the corpuscle samples were always examined the same day to avoid the effects of bacterial contamination. I have since found stated in the literature that corpuscles may be desiccated[8] or even heated to 100° C.[15] without diminution of the iso-agglutinogen content. An effect of bacterial contamination is hæmolysis of the corpuscles, but it seems very probable that sufficient asepsis or antisepsis could be obtained in the light of the stability of the agglutinogen to secure the preservation of corpuscle samples over a sufficient period to render very simple the routine examination at institutions situated similarly to Palm Island.

Again in this examination I endeavoured to use sera as fresh as possible. At the commencement of the series only were some samples of serum more than thirty-six hours old. The serum used at the commencement was prepared in Townsville on February 3, placed in sterile tubes and sealed, opened when required and used on the fourth and fifth. Compared on the seventh with serum then only thirty hours old, they showed in every case (nineteen samples were tested with both sets) exactly the same reaction. On another occasion serum thirty hours old was compared with serum prepared the same day in its reaction on twenty specimens shown to belong to Groups II., III. and IV. by the fresh serum. Again no qualitative discrepancy in the result was found. On a third occasion eight samples diagnosed as belonging to Group I. by serum thirty hours old were retested by fresh serum; no trace of agglutination was seen even under the microscope. In some cases, it must be admitted, it seemed that the older serum required longer to produce the same degree of agglutination, but the total result was about the same.

An examination of literature yields several references to the stability of the agglutinins. Sanford[2] says: "It is well known that the iso-agglutinins in human serum are thermo-stable" and records his

TABLE I.—FREQUENCY OF VARIOUS GROUPS (CLASSIFICATION OF JANSKY).

Author.	Group I. Agglutinogen *O*		Group II. Agglutinogen *A*		Group III. Agglutinogen *B*		Group IV. Agglutinogen *AB*		All Groups	Biochemical Index $\frac{AB+A}{AB+B}$
	No.	%	No.	%	No.	%	No.	%		
Tebbutt and McConnel ..	105	55·0	73	38·2	11	5·8	2	1·1	191	5·7
Lee	227	60·3	120	31·7	24	6·4	6	1·6	377	4·2
All Authors ..	332	58·5	193	33·9	35	6·2	8	1·4	568	4·67

"Biochemical" Index.—The first name assigned to this index was apparently that of the Hirschfeld[3]: "Biochemical Race Index." This has become shortened to "Biochemical Index" or simply to "Race Index" (Kossovitch,[10] *"Indice des Races"*). Tebbutt[9] suggests the much more appropriate name of "Isoagglutinin Index."

experiments to show that they resist also desiccation. Bais and Verhoef, working in the tropical climate of Sumatra and Java, state[14]: "The test sera . . . were . . . replaced by a fresh stock each week," though they do not say whether they were kept in an ice box or had some preservative, such as tricresol added. Vincent[1] used tricresol 0·25% "because it is desirable to keep the serum sterile," though he records the observation that there was "no apparent diminution in the activity of serum one year old which showed a moderate degree of contamination."

Results.

Three hundred and seventy-seven aborigines in all were examined, ten at the Townsville General Hospital, having come there from the Palms and the remainder at the Palms. Those examined at the Settlement were sent up by Superintendent as being full blooded aborigines and any of those who showed "frizzy" hair or absence of the flattened nose or in general appearance gave the impression of not being full blooded aborigines, were not examined. The names of those who on examination proved to belong to Groups II., III. or IV. were read over to one of the staff who stated them to be all full blooded aborigines to the best of his knowledge. Beyond the limits of these precautions I cannot vouch for the purity of the subjects, since records when available are always to be taken with reservation in cases like this, where parentage is often a doubtful question.

The results of examination are given in Table I., in which appear also the results previously obtained by Tebbutt and McConnel.[8] [9]

The results obtained in the two sets of investigations agree in placing the Australian aboriginal very high up in the series of racial "biochemical" indices, though they differ considerably in the actual magnitude of the index. When the indices for various races are considered, it will be seen that in the list there given only one race lies above the position assigned by Tebbutt and McConnel's results, the North American Indian, who has an index of 9·2. Between 5·7 and 4·2 lie three European races, the Angles (5·35), the Alpine race (4·95) and the English (4·55); all the other races so far investigated (as far as I could find in the available literature) lie below 4·2, reaching as low as 0·5.

As far as Tebbutt and McConnel's results for aborigines are concerned there can be no reasonable doubt as to their authenticity, because all their findings were confirmed by cross-agglutination experiments.[16] My examination on the other hand, was made by the straight-out method of Moss which is open to two objections: (i.) That there is no check on the actual recording of the results, (ii.) that errors in technique interfering with the power of the serum to react are less easily noticed. The major errors affecting a whole batch are easily noted. With regard to the first, all due care was exercised and the slides checked over against the entries on the record sheets before their removal. With regard to the second also, the greatest care was exercised and a regular methodical routine used throughout. As some proof of the reliability of the results the retests may be cited. Thirty-seven out of forty-eight subjects whose blood was found to belong to Group I. on two days, were re-examined (the remaining eleven being unavailable) and all the samples yielded exactly the same results. Of the thirty-nine comparative tests (see above) made on the same samples with two sets of serum, not one showed any discrepancy. With careful working the number of such errors must be small and if they do occur, they will follow the laws of chance and should not affect the index, which is a ratio between two frequencies determined at the same examination.

It seems that the difference between the results may be referred to the geographical positions of the subjects examined on the two occasions. Quite

TABLE II.

Germans.		Japanese.		Koreans.		Chinese.		Russians.	
Heidelberg ..	3·12	South	1·82	North	1·05	Central	1·42	Central	1·5
Baltic	3·02	Middle	1·68	Middle	1·26	South	1·08	Siberia	1·26
Leipzig	2·04	North	1·58			Pekin	0·50	Perm	1·07
Berlin	2·0							Ukraine	1·05
Munich	2·0								

frequently within the same race (this will be referred to again later) I find that the index varies with the geographical position of the people examined (by "position" is meant their place of birth; migrants, of course, cannot be expected to show an association of grouping with place of residence), as Table II. will show.

It may be that such is the case with the Australian aboriginal. Tebbutt and McConnel give the origin of those of their subjects for whom records were available[9]: Cape York 39 (26%), north Queensland 21 (14%), central Queensland 36 (24%), southern Queensland 53 (36%). Thus well over half (including the nineteen from New South Wales) came from well south of Townsville. An analysis of the origins of those examined by me, on the other hand, yields the following figures:

Total number examined: 377.

Total number for whom records were available: 358.

TABLE III.—DISSECTION OF RECORDS.

Peninsula (south to Cairns and district inclusive)	163
Gulf	55
North-west Queensland	28
Coast (south of Cairns to Townsville inclusive)	71
Coast (south of Townsville to Mackay inclusive)	24
Northern (exact location unknown)	17
Total	358

These records were furnished by courtesy of Mr. Hoffmann, of the Settlement.

I may say, therefore, that the series of aborigines examined were wholly from north Queensland. Hence I may justly conclude that I have here a further indication of variation of index within a race, with the geographical origin of the group examined. I shall see later that this is only a continuation of a general variation of index with geographical position. I may notice here the recent publication of results obtained by Cleland[20] on South Australian aborigines. Out of one hundred and one examined forty-six belonged to Group I. and fifty-five to Group II. He found none belonging to Group III. or IV. This is the extreme end of the gradient that I suggest exists.

Variation on Successive Days.

The figures given in Table IV. showing the variation in results from day to day during the

TABLE IV.

Date.	Percentage.				Biochemical Index.	
	I.	II.	III.	IV.	For Day.	For Total to Date.
Jan. 28	50	40	10	0	4·0	4·0
Feb. 4	66	33	0	0	—	9·0
Feb. 5	56	33	9	0	2·8	3·8
Feb. 6	70	22	6	4	2·6	3·3
Feb. 7	70	20	5	0	4·0	3·3
Feb. 8	52	38	6	2	5·0	3·75
Feb. 12	57	39	4	0	9·0	4·3
Feb. 13	68	24	4	2	4·3	4·3
Feb. 14	52	40	8	4	3·7	4·25
Feb. 15	58	36	6	2	4·75	4·3
Feb. 16	48	20	8	0	2·5	4·2

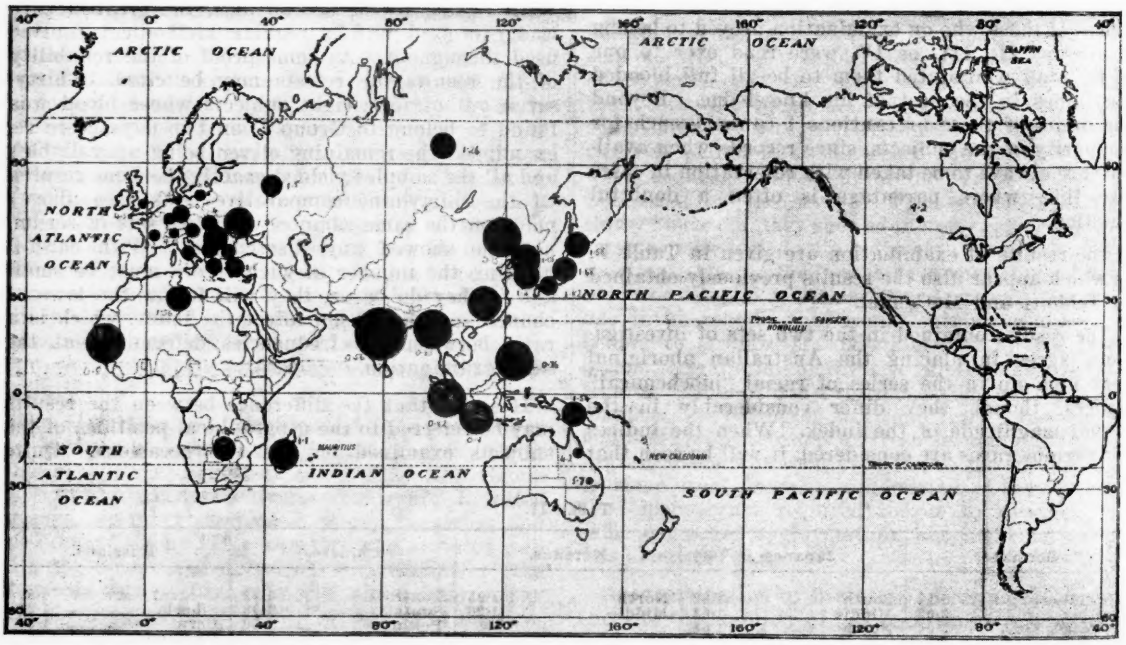

FIGURE I.

examination may serve a double purpose; firstly to show the unreliability of small numbers and secondly to give additional support to the authenticity of the index, in that it remains stable over the last five days of the examination, when the numbers become appreciable.

Comparison Between Calculated and Observed Frequency of Groups I. and IV.

In their article in *The Lancet* the two Doctors Hirschfeld give a method of calculating from the observed frequencies of Groups II., III. and IV. the probable frequencies of Groups I. and IV. (*O* and *AB* respectively). Unfortunately, the method they set out for calculating that of Group IV. is incorrect and this error has been perpetuated by those few workers who have endeavoured to calculate the probable frequencies.[1] The error is best illustrated by setting out the correct method and then pointing out where that used by Doctors Hirschfeld is incorrect.

[1] In his paper read to the Pan-Pacific Science Congress Tebbutt included a calculation of this nature which was made by the correct method. The publication[(9)] quoted in this article is a summary of the proceedings.

I shall take for convenience the same figures (English) that Doctors Hirschfeld employed in their considerations. It is necessary to note that the classification used by Doctors Hirschfeld is Moss's, while that used here, in common with American serologists, is Jansky's. According to the Jansky classification the figures are:

TABLE V.

I.	II.	III.	IV.
(*O*)	(*A*)	(*B*)	(*A B*)
46·4	43·4	7·2	3·0

The *A* factor occurs in 43·4 + 3·0 = 46·4% of the population. That is out of any group selected at random (as far as the possession of *A* is concerned) out of this population, 46·4% may be expected to contain the *A* factor in their corpuscles.

Similarly, the *B* factor occurs in 7·2 + 3·0 = 10·2% of the population.

Now, if the possession of *A* factor and the possession of *B* factor are absolutely independent of each other, it may be expected that 10·2% of those who

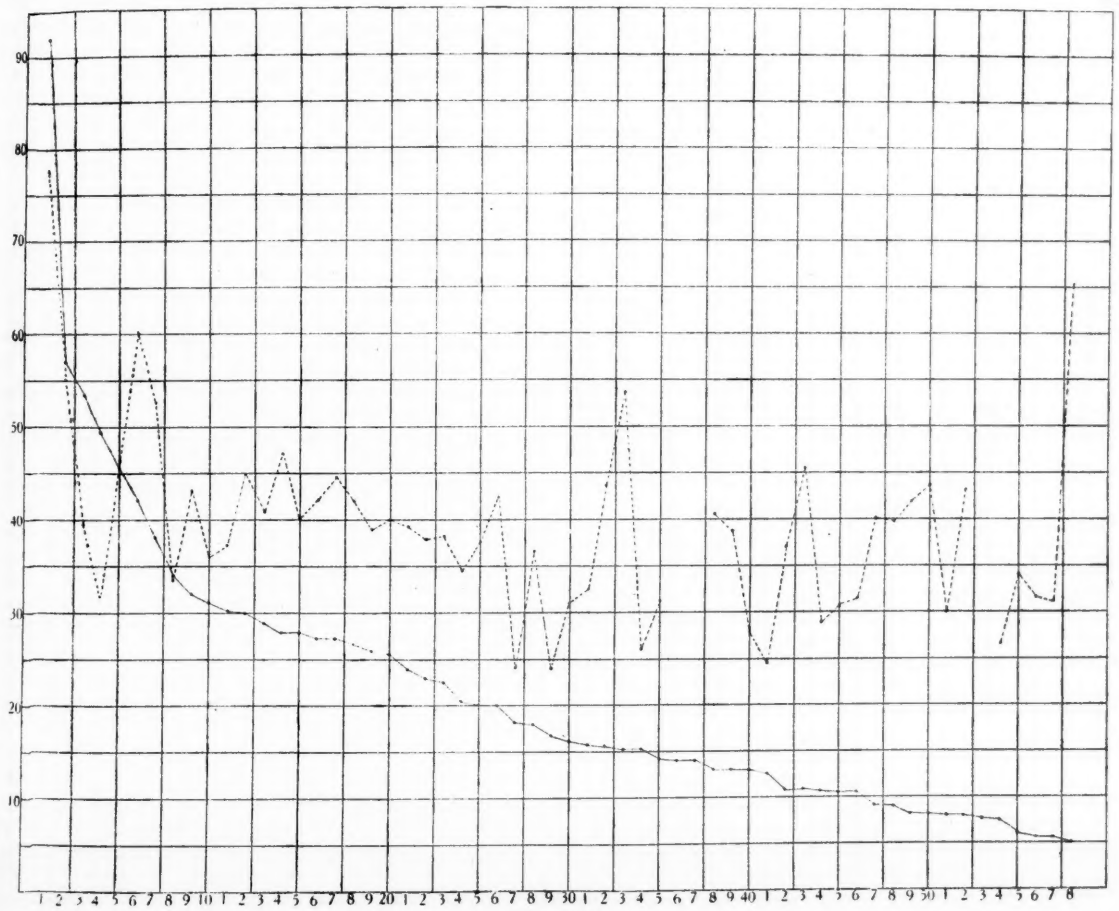

FIGURE II.

contain *A* factor, contain also the *B* factor, because this group of people, although selected to contain *A*, have been selected quite at random as far as the possession of *B* is concerned. That is to say, that of the 46·4% of people containing *A* factor, I should expect 10·2% to harbour *A* and *B* together, that is, to belong to Group IV.

Therefore, the probable frequency of Group IV. in this population is $\frac{46{\cdot}4 \times 10{\cdot}2}{100} = 4{\cdot}7\%$.

By a similar process the probable frequency of Group I. may be calculated in the following manner:

The *A* factor occurs in 46·4%; therefore the non-*A* factor occurs in 100 – 46·4 = 53·6%.

Similarly the non-*B* factor occurs in 100 – 10·2 = 89·8%; therefore the probable frequency of non-*A* and non-*B* together (that is Group I.) is $\frac{53{\cdot}6 \times 89{\cdot}8}{100}$ = 48·1%.

I may now point out the error in the Hirschfeld method for the first part of this calculation. Instead of multiplying the *A* factor frequency by the *B* factor frequency and dividing by 100, they multiplied Group *A* (Group II.) frequency by Group *B* (Group III.) frequency and divided by 100. Now clearly this cannot be allowed for two reasons. (1) Group *A* does not represent the total frequency of the *A* factor, it is less than that frequency; the same obtains in regard to the *B* factor. (2) Group *A* is specially selected as regards the *B* factor. It is selected as not possessing the *B* factor, a negative relation, still a relation. What the method attempts to do, in other words, is to calculate how many Group II. people will also belong to Group III., when all the time the two groups are mutually exclusive. The error is nothing more than one in fundamental deductive logic, though that logic happens to be applied to mathematical considerations. I do not doubt that the Hirschfelds would have realized this mistake if only they had used the term *A* factor to distinguish the total frequency of the agglutinogen *A* from the frequency of people who possessed the agglutinogen *A* in isolation (Group II.); and similarly for *B*.

Strangely enough, they rectified their mistake when calculating for Group I., their method being that outlined above. It is a great pity that this error has been allowed to pass for so long.

Geographical Distribution of the Biochemical Index.

In going through the available literature on the question of the distribution of biochemical index

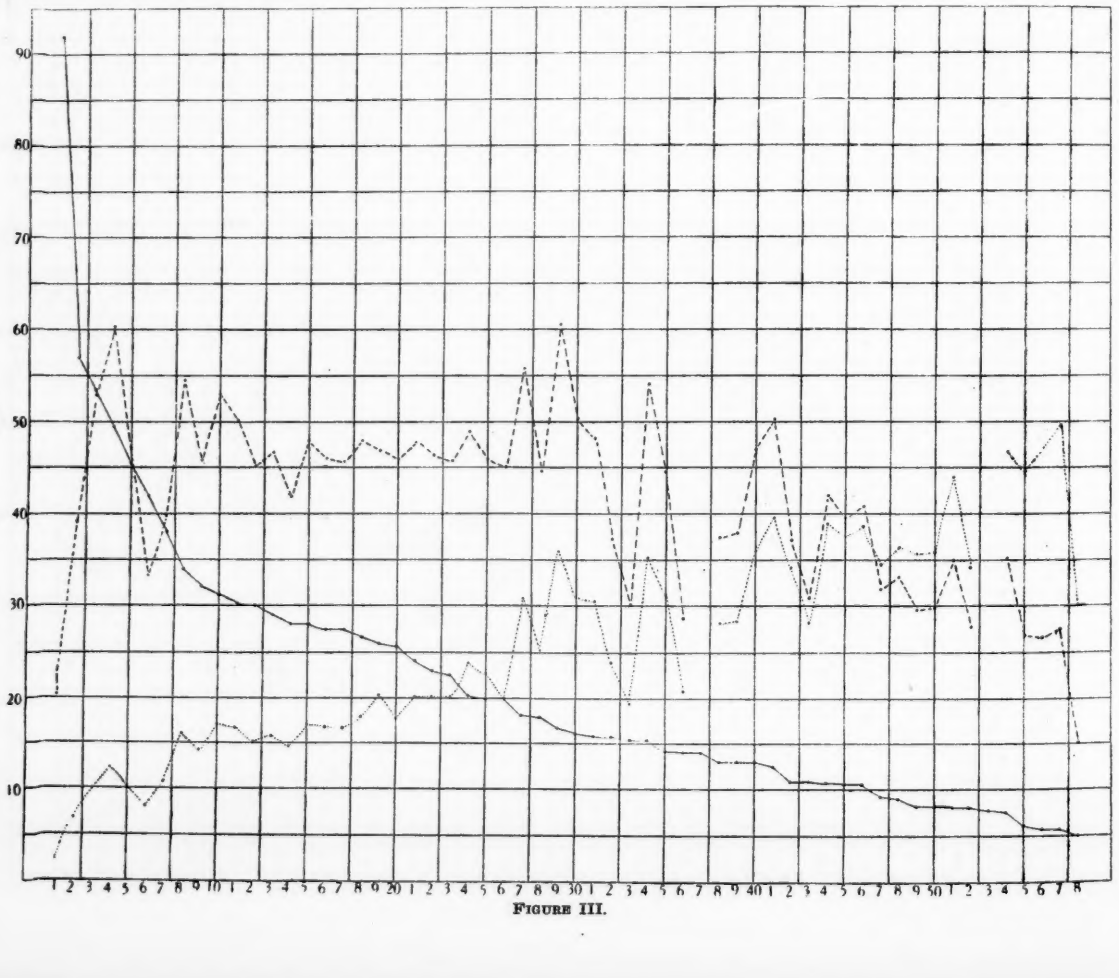

FIGURE III.

through the races of the world I found numerous references to the outstanding feature that from Western Europe eastwards to India there is a steady falling off in the index, followed by a tendency to rise again in Indo-China. But I found only one attempt at a statistical collection of the published figures, namely that given by Steffan.[18] This list has some important omissions, Australian aborigines, Melanesians, Bantus and certain other figures have since been published. This collection, moreover, contains some inaccuracies. I have, therefore, thought it wise to make as complete a collection as possible before trying to follow the geographical distribution of the index. The figures given in Table VI. have been culled from the available literature and in submitting them I wish to state that I do not claim for it completeness to date, since both time and the literature available have been necessarily limited. I invite all interested to criticize, correct and rectify omissions as soon as possible. The figures which are especially wanting, are those for South American natives, Maoris, Fijians, South African bushmen, Norwegians, Swedes and Eskimos. In several places I have found inaccuracies both in quotations and in calculation and these I have endeavoured to rectify. The calculations for probable frequencies of Group IV. have been made according to the method given above and disagree, therefore, from previously published figures. Discussion is especially invited on the question, as to whether the differences between observed and calculated frequencies of Group I. and Group IV. are compatible with the idea that the *A* and *B* factors are independently heritable.

Figure I. shows the distribution of the biochemical index. It will be seen that India forms the centre of a cone-like depression with an index of 0·56 and that as we pass out from it the figures for the most part show a steady gradient, but more especially to the east and west, until the extremes of 5·35 in north-west Europe and 5·7 in Australia and 9·2 in North America are reached. If instead of symbols the figures were plotted, it would be found that this appearance is very well marked. With symbols the smaller variations cannot be well shown. This coincidence of variation in the index with geographical distribution may be tentatively referred to two sets of causes, the one to be found in geographical factors acting as such upon the hereditary transmission of the *A* and *B* factors from parent to offspring and thus modifying the index of the race; the other to be found in considerations of the place of origin of man and his subsequent migrations. The first suggestion has apparently been dismissed up to the present as being unlikely, but it seems that this decision is somewhat too summary. The idea, it would seem, it at least worthy of a little more consideration.

As regards the second suggestion it would seem from this that at one time in the racial history of man there was a "biochemical" race *A*, containing only the *A* agglutinogen in the corpuscles, of practically universal distribution; later a second "biochemical" race *B* arose in India or thereabouts and gradually infused into the race *A*. In that way would be produced the high percentage of *B* individuals, leading to a low index in the Indian region, while a gradual decrease in percentage of *B* individuals with a corresponding increase in *A* individuals and in the index, would be met with the further we get away from that region. The point of origin of the first race *A*, must be left an open question, as far as the evidence given by blood groups is concerned; all that I can say is that at the time of the supposed origin of the *B* race the *A* race was apparently universally distributed.

To return to the two series of investigations on the Australian aborigines, it will be noticed that the direction of the gradient is exactly that which would be expected, an increase from north to south. Cleland's results confirm this very strikingly.[20] I should very much like to have measurements made on the aborigines of north-west Australia and of the Northern Territory.

Figure II. gives the frequency of Group I. as compared with descending biochemical index, while Figure III. gives the frequency of *A* and *B* factors when the races are arranged in order of descending biochemical index. It is hoped that these figures will give rise to some discussion amongst those interested and then this will lead to the disclosure of some noteworthy facts deducible therefrom. It cannot be too strongly insisted that truth must be sought in this line by cooperation between anthropological and serological methods.

In conclusion, I should like to urge the undertaking of numerous measurements on well-defined races in all parts of the world and a collection of all these figures for anthropological and statistical investigation. More especially does the duty devolve upon us, as Australians, of making as many investigations of this nature on the Australian aborigine as possible, while the opportunity exists. Let us not be forced to admit that we neglect those features of scientific interest for which our country is unique. The aborigine is undoubtedly a dying race and in very few generations the opportunity will be gone.

Acknowledgments.

This work was undertaken at the Institute of Tropical Medicine, Townsville and my thanks are due to the Division of Tropical Hygiene, Commonwealth Department of Health, for bearing the expenses incurred in the investigation and for the loan of all materials employed. Amongst the staff, Dr. Heydon especially has earned my gratitude; it was he who led me to make this research; I thank him for his indispensable help and suggestion. throughout. Thanks are also due to Dr. Broben for assistance in German translation. The members of the staff of the Aboriginal Settlement were of great assistance and especially Mr. Hoffmann by his interest in the work. My thanks are also due to Dr. A. H. Tebbutt for his kindly suggestion and for further references.

TABLE VI.

Graph No.	People.	Workers.	Reference No.	No. Exam.	Percentage. O. I.	Percentage. A. II.	Percentage. B. III.	Percentage. AB. IV.	Group. I % obs. (O_1.)	Group. I % calc. (C_1.)	Group IV. % obs. (O_4.)	Group IV. % calc. (C_4.)	Percentage. A. II. + IV.	Percentage. B. III. + IV.	Bloch. Index.
1	North American Indians ..	C., Dl.	12	862	77·7	20·2	2·1	0·1	77·7	78·1	0·1	0·4	20·3	2·2	9·2[1]
2	Australian aborigines	T., Mc.	9	191	55·0	38·2	5·8	1·1	55·0	56·5	1·1	2·7	39·3	6·9	5·7
3	Angles	St., Sh.	18	253	39·5	50·6	7·5	2·4	39·5	42·3	2·4	5·25	53·0	9·9	5·35
4	*Homo alpinus* (Peterstal) ..	Kt., St.	18	502	31·8	56·0	7·8	4·4	31·8	34·8	4·4	7·4	60·4	12·2	4·95
5	English	H.	3	500	46·4	43·4	7·2	3·0	46·4	47·9	3·1	4·7	46·4	10·2	4·55
6	Australian aborigines	Lee	..	377	60·3	31·7	6·4	1·6	60·3	61·3	1·6	2·7	33·3	8·0	4·2
7	Australian whites	T., Mc.	9	1,176	52·6	36·8	7·5	3·1	52·6	53·7	3·1	4·2	39·9	10·6	3·8
8	German settlers in Transylvania	Mu.	18	301	33·5	50·5	12·0	4·0	33·5	38·2	4·0	8·7	54·5	16·0	3·4
9	French	H.	3	500	43·2	42·6	11·2	3·0	43·2	46·6	3·0	6·4	45·6	14·2	3·2
10	Germans (Heidelberg)[2] ..	v. D., H.	18	[3]500[10]	36·0	47·3	11·3	5·7	36·0	39·0	5·7	9·0	53·0	17·0	3·12[2]
11	Germans on the Baltic	St.	18	1,353	37·4	45·7	12·1	4·5	37·4	41·5	4·5	8·3	50·2	16·6	3·02
12	United States	After Vz.	18	?	45·0	40·0	10·0	5·0	45·0	46·8	5·0	6·75	45·0	15·0	3·0
13	German settlers in Hungary	Vz.	18	476	40·8	43·5	12·6	3·1	40·8	45·0	3·1	7·3	46·6	15·7	2·9
14	Italians	H.	3	500	47·2	38·0	11·0	3·8	47·2	49·5	3·8	6·2	41·8	14·8	2·8
15	Germans[3]	v. D., H.	3	[3]500	40·0	43·0	12·0	5·0	40·0	43·1	5·0	8·2	48·0	17·0	2·8[3]
16	Berlin Jews	Sf., Ze.	18	230	42·1	41·1	11·9	4·9	42·1	44·9	4·9	7·7	46·0	16·8	2·74
17	United States[11]	Hk., Mo., Ot.	4	456	44·7	38·7	9·7	7·0	44·7	45·2	7·0	7·6	45·7	16·7	2·74
18	Austrians	L.	3	?	42·0	40·0	10·0	8·0	42·0	42·6	8·0	8·6	48·0	18·0	2·67
19	Bulgarians	H.	3	500	39·0	40·6	14·2	6·2	39·0	42·3	6·2	9·5	46·8	20·4	2·6
20	Banat Germans	Mu.	18	414	40·0	42·1	14·0	3·9	40·0	44·3	3·9	8·2	46·0	17·9	2·57
21	Czechos	Kv.	19	218	39·2	40·0	12·4	7·8	39·2	41·7	7·8	9·7	47·8	20·2	2·4
22	Serbs	H.	3	500	38·0	41·8	15·6	4·6	38·0	42·7	4·6	9·4	46·4	20·2	2·3[4]
23	Greeks	H.	3	500	38·2	41·6	16·2	4·0	38·2	43·4	4·0	9·2	45·6	20·2	2·26[5]
24	Leipzig	Sk.	18	1,000	34·5	41·5	16·5	7·5	34·5	38·8	7·5	11·8	49·0	24·0	2·04
25	Berlin	Sf., Ze.	18	750	37·8	39·4	16·4	6·4	37·8	41·8	6·4	10·4	45·8	22·8	2·0
26	Munich	D., S.	4	155	42·6	37·4	17·4	2·6	42·6	48·0	2·6	8·0	40·0	20·0	2·0
27	South Japanese	F.	10	170	24·1	45·3	20·2	10·6	24·1	30·5	10·6	17·2	55·9	30·8	1·82
28	Turks	H.	3	500	36·8	38·0	18·6	6·6	36·8	41·5	6·6	11·2	44·6	25·2	1·8
29	Japanese (middle)	F.	10	353	24·0	40·5	16·0	20·0	24·0	25·3	20·0	21·8	60·5	36·0	1·68
30	Hungarians	Vz.	18	1,500	31·0	38·0	18·8	12·2	31·0	34·4	12·2	15·6	50·2	31·0	1·62
31	North Japanese	F.	10	151	32·5	37·0	19·2	11·3	32·5	35·9	11·3	14·7	48·3	30·5	1·58
32	Arabs	H.	3	500	43·6	32·4	19·0	5·0	43·6	47·5	5·0	9·0	37·4	24·0	1·56
33	Melanesians	Hy., M.	17	753	53·7	26·8	16·3	3·2	53·7	56·3	3·2	5·8	30·0	19·5	1·54
34	Roumanian Jews	Mu.	18	211	26·1	38·8	19·8	15·3	26·1	29·8	15·3	19·0	54·1	35·1	1·54
35	Central Chinese	F.	10	45	31·1	37·8	24·4	6·7	31·1	38·2	6·7	13·8	44·5	31·1	1·42
36	Bantus	P.	7	250	..	..	..	..	..	..	..	..	28·8	20·8	1·4
37	American Negroes	Hd.	18	..	49·0	26·9	18·5	5·5	49·0	51·4	5·5	7·8	32·4	24·0	1·4
38	Russians	H.	3	1,000	40·7	31·2	21·8	6·3	40·7	44·9	6·3	10·5	37·5	28·1	1·3[6]
39	Jews (Monastir)	H.	3	500	38·8	33·0	23·2	5·0	38·8	44·5	5·0	10·7	38·0	28·2	1·3
40	Chinese	K., Li.	12	100	28·0	36·0	25·0	11·0	28·0	33·9	11·0	16·9	47·0	36·0	1·3
41	Koreans (middle)	F.	10	179	24·6	35·8	25·1	14·5	24·6	30·0	14·5	19·9	50·3	39·6	1·26
42	South Chinese	F.	10	35	37·1	28·6	25·7	8·6	37·1	41·4	8·6	12·8	37·2	34·3	1·08[7]
43	Malagasies	H.	3	400	45·5	26·2	23·7	4·5	45·5	49·5	4·5	8·6	30·7	28·2	1·09
44	Chinese (mixed)	C., Dl.	12	111	29·0	32·0	29·0	10·0	29·0	35·4	10·0	16·4	42·0	39·0	1·07
45	Chinese (all)	F. aft. C., Di.	12	?	30·9	31·2	29·3	8·4	30·9	37·6	8·4	14·9	39·6	37·7	1·05
46	North Koreans	F.	10	184	31·5	29·9	27·7	10·9	31·5	36·4	10·9	15·7	40·8	38·6	1·05
47	Chinese (mixed)	B., V.	14	592	40·2	25·0	27·6	6·9	40·2	44·2	6·9	11·0	31·9	34·5	0·92
48	Javanese	B., V.	14	1,346	39·9	25·7	29·0	7·4	39·9	44·2	7·4	12·0	33·1	36·4	0·9
49	Annamese	H.	3	500	42·0	22·4	28·4	7·2	42·0	45·3	7·2	10·5	29·6	35·6	0·83
50	Sumatrans	B., V.	14	546	43·7	23·0	29·0	6·7	43·7	48·8	6·7	10·6	29·7	35·7	0·82[8]
51	Chinese[12]	Li., W.	3[1]	1,000	30·7	25·1	34·2	10·0	30·0	36·4	10·0	15·4	35·0	44·0	0·8
52	Senegalese	H.	3	500	43·2	22·6	29·2	5·0	43·2	47·6	5·0	9·4	27·6	34·2	0·8
53	Filipinos	Cb., Wd.	7[1]	132	64·7	14·7	19·2	1·0	64·7	66·9	1·0	3·2	15·7	20·2	0·76
54	Manchus	F.	10	199	26·6	26·6	38·2	8·5	26·6	34·6	8·5	16·4	35·1	46·7	0·75
55	Gipsies in Hungary	Vz.	18	385	34·2	21·1	38·9	5·8	34·2	40·4	5·8	12·0	26·9	44·7	0·6
56	Cingalese	B., V.	18	79	31·6	20·3	40·8	6·3	31·6	38·8	6·3	12·5	26·6	47·1	0·56
57	Indians	H.	3	1,000	31·3	19·0	41·2	8·5	31·3	36·5	8·5	13·7	27·5	49·7	0·56
58	Pekin Chinese	H.-S.-L., St.	18	427	65·4	4·7	19·7	10·3	65·4	59·5	10·3	4·5	15·0	30·0	0·50[9]

For footnotes see the following page.

FOOTNOTES TO TABLE VI.

[1] Steffan gives 8·8 which does not agree with the percentages he quotes, which are those of Coca and Diebert's second group of 862 students.

[2, 3] Hirschfeld quotes the figures of von Dungern and Hirschfeld given in No. 15 from memory. It is not clear whether the figures given by Steffan in No. 10 as those obtained by von Dungern and Hirschfeld are for the same series, rectified by consulting the original figures, or whether they are for a separate series.

[4, 5] Hirschfeld gives 2·5 for each, which does not agree with their published percentages.

[6] Hirschfeld analyses this series as regards the occurrence of *A* and *B* factors as follows:

Region.	Percentage.		Total.
	A.	B.	
Central Russia ..	37·6	25·2	400
Siberia	36·5	29·0	321
Ukraine	35·1	33·3	111
Perm, Vologda, etc.	36·8	34·5	84

This gives indices of 1·5, 1·26, 1·05 and 1·07 respectively.

[7] Fukamachi's published figure is 1·18 which is incorrect for his frequencies.

[8] The percentages add up to 102·2. This latitude is too wide. A similar discrepancy exists in his results for the Javanese (No. 47).

[9] These results are rather unexpected. Confirmation would be welcomed.

[10] Ottenberg[4] gives 348 as the number examined.

[11] The figures given by Ottenberg[4] as the results of his own previously unpublished work, together with the figures of Moss and Hektoen, have been averaged in this series. The authors do not expressly state whether the subjects were whites.

[12] These were not all full-bloods.

EXPLANATORY NOTES TO TABLE.

Workers' names are indicated by the following abbreviations:

B., Bais.
C., Coca.
Cb., Cabrera.
D., Descatelle.
v. D., von Dungern.
Di., Diebert.
F., Fukamachi.
H., Hirschfeld.
Hd., Henderson.
Hk., Hektoen.
H.S.L., Hung See Liu.
Hy., Heydon.
K., Kilgoe.
Kt., Ketterer.
Kv., Kossovitch.
L., Landsteiner.
Li., Liu.
M., Murphy.
Mo., Moss.
Mc., McConnel.
Mu., Manuila.
Ot., Ottenberg.
P., Pirie.
S., Sturli.
Sf., Shiff.
Sh., Schutze.
Sk., Sucker.
St., Steffan.
T., Tebbutt.
V., Verhoef.
Vz., Verzar.
W., Wang.
Wd., Wade.
Z., Zeigler.

APPENDIX TO TABLE.

Since the table given above was compiled and the graphs drawn, the author has seen a reprint of an article by Hirschfeld in *"L'Anthropologie,"* Volume XXIX., 1918-19, page 505. This contains a dissection of the usually quoted figures according to subraces. Since it seems to me that the averaging of subraces is seldom justifiable in a consideration of the biochemical index in its distribution and since time is not available for a revision of the table, graphs and map, I thought it best to reproduce these figures in the form of an appendix to this table. It must be noticed, that the dissected figures appearing hereunder are not shown in any of the graphs or in the map contained in the body of the article.

ENGLISH.

TABLE V.

Sub-Race.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
English	180	44·6	180	44·6	31	7·7	12	3·0	403	4·4
Welsh	12	—	11	—	3	—	3	—	29	—
Scotch	29	—	21	—	2	—	0	—	52	—
Irish	11	—	5	—	0	—	0	—	16	—

FRENCH.

The numbers in the separate provinces too small to be given.

ITALIANS.

TABLE VI.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
North (Piedmont Lombardy, Venice)	90	44·8	84	41·8	20	9·9	7	3·5	201	3·4
Central	43	54·4	27	34·3	4	5·0	5	6·3	79	—
South	103	46·8	79	36·0	31	14·0	7	3·2	220	2·3

SERBS.

Differentiation into provinces revealed no great differences.

GREEKS.

TABLE VII.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
Asia-Minor	48	31·8	71	47·0	26	17·0	6	4·0	151	2·4
Greece	50	38·5	55	47·2	21	16·6	4	3·0	130	2·6
Thrace	35	—	24	—	12	—	6	—	77	—
Archipelago	26	—	25	—	8	—	1	—	60	—
Crete	15	—	20	—	6	—	1	—	42	—
Macedonia	14	—	10	—	6	—	1	—	31	—
Epire	3	—	3	—	2	—	1	—	9	—

BULGARIANS.

There is perhaps a greater frequency of *B* around Varna and the shore of the Black Sea.

RUSSIANS.

The data are given as a note to Table VI. and in the map.

ARABS.

TABLE VIII.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
Tunis	100	50·0	50	25·0	39	19·5	11	5·5	200	1·2
Algeria	118	39·0	112	37·6	56	18·6	14	4·6	300	1·8

MALAGASIES.

Of 400 examined 266 were Hovas. The rest were scattered among several races. The figures for the Hovas are given in Table IX.

TABLE IX.

	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
	120	45·5	73	27·5	61	22·5	12	4·5	266	1·1

NEGROES.

Of the 500 examined 238 were from Senegal, 81 Bambaras and the rest very much assorted in race. The figures for these two main groups are given in Table X.

TABLE X.

Race.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.
Senegal	110	46·2	50	21·0	71	29·7	7	2·9	238	0·7
Bambaras	29	35·8	20	24·7	32	6·0	6	7·4	81	—

INDO-CHINESE.

Of the 500 examined 397 were from Tonkin. The index for these is 0·84.

INDIANS.

Two series of 500 were examined, but Hirschfeld suspects that the second series is not as reliable as the first, because the blood samples were not tested until the day following their collection. Only the provinces from which more than one hundred were examined are given below.

TABLE XI.

Origin.	Groups. I. No.	I. %	II. No.	II. %	III. No.	III. %	IV. No.	IV. %	Total.	B.I.	Series.
United Provinces	23	22·3	18	17·4	52	50·5	10	9·7	103	0·45	1st
Central ,,	67	37·4	43	24·0	62	34·6	7	3·9	179	0·73	2nd
Punjab	63	24·0	41	15·6	121	46·1	37	14·1	262	0·50	1st

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POINTS FROM PRACTICE: DIAGNOSTIC AND PROGNOSTIC.[1]

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As an example of how we would all like to diagnose conditions I often think of a patient who suffered from debility and diarrhœa of three weeks' duration.

A doctor, admitting the patient to hospital, ordered catechu and opium and starch and opium enemata, but the treatment failed. A house surgeon then examined digitally the interior of the rectum and found it to be full of hard masses of fæces which were causing the diarrhœa. So the condition was really constipation. The bowel condition was soon cleared up. The patient's persistent weakness was now vaguely supposed to be due to the heart and he was having slight attacks of dyspnœa. After about a fortnight the diagnosis was made the very moment a new honorary stepped inside the ward. He said: "There's a gander cough! Which is the patient with the aneurysm?" He had noted the very resonant, brassy cough which is produced when the lumen of the trachea is encroached upon by a mediastinal tumour or by an aneurysm, particularly an aneurysm of the transverse arch of the aorta.

An aneurysm in that situation is quite devoid of physical signs, but causes attacks of dyspnœa with cough and stridor, all due to pressure on the trachea.

How convenient it is to have once seen an ulceration on the chest of a diabetic or to be acquainted with the fact that *herpes zoster* may be caused by arsenic in the system or to know that dizziness may be the only symptom of an otherwise untraceable abscess of the scalp, the only other signs being feverishness soon passing off and perhaps a big white tongue.

Pyelitis is a diagnosis that does not always give final satisfaction, even when the diagnosis is correct. It is not nice to treat a patient for some years for attacks of what you call pyelitis with copious pus in the urine and to be so sure of the diagnosis that you forget to have the kidney examined by X rays for stone. I believe this mistake is fairly common.

Perhaps another pyelitis patient after several attacks will mention that he or she suffers from sneezing and the nose specialist will discover what is at the bottom of the pyelitis, namely, pus in the maxillary antrum. Or in another case of pyelitis the testing of the urine for sugar may give a positive result and you will then have this interesting problem: Is the glycosuria secondary to the pyelitis, the pyelitis having lowered the renal threshold for sugar just the same as carbuncle or furunculosis may do or conversely is the pyelitis a complication of diabetes, just as other inflammatory conditions such as *otitis media* may complicate diabetes?

As to diabetes itself it is remarkable that I have discovered it in two patients who complained of feeling run down and of losing weight and who had just recently consulted other practitioners for this

[1] Read at a meeting of the Queensland Branch of the British Medical Association on May 7, 1926.

without the ailment having been discovered. The clue to the diagnosis of several diabetes cases has been: "Run down, legs very tired, losing weight," especially "legs very tired."

Acute appendicitis is not necessarily accompanied by retching or vomiting, even if the patient eats, but particularly is it true in regard to some patients who cease eating when the attack comes on. Such a patient, if not operated on, runs just as much risk as one who vomits.

In regard to pneumonia, an initial chill is said to be more common in pneumonia than in any other acute disease; the temperature taken during this initial feeling of coldness often shows that the fever has already begun. Not always, though. The temperature at the onset may be found by you to be 35·6° C. (96° F.), even though the patient is in a warm bed. He looks miserably cold. There may be even then a slight suggestion of stitch in the side. You exclude very carefully any other causes of collapse temperatures, but make sure by coming back in an hour's time, when you find him looking more like a patient with pneumonia should. His temperature is now 39·4° C. (103° F.) say, possibly there is a suspicion of slight friction and after a day or two the usual complete signs and symptoms of pneumonia appear.

A patient is under observation for something else and the temperature shoots up practically at once to 40° C. (104° F.). Nothing is complained of, except perhaps a little headache and malaise. (That same description so far might apply to a hospital patient who is contracting smallpox.) With one particular pneumonia patient the temperature came down within twenty-four hours—a one-day pneumonia. Plain signs of consolidation appeared about twenty-four hours later without any recurrence of feverishness. The two correct diagnoses in this case and likewise in the smallpox case respectively were made straight away at first sight of the temperature chart by excellent guesswork.

Notice that headache may be the only complaint of a patient with pneumonia and also the respiratory rate may be almost normal. Hence for a day or two or even more, while waiting for the chest signs to come out, one often wrongly suspects that the condition is typhoid or in these days influenza. Delirium may occur for two days or so after the crisis and though it is worrying, it is not of bad omen, if your patient seems reasonably strong. Death may occur forty-eight hours or more after the crisis, the patient, a man, say, of sixty years, getting a far away look and dying of heart failure just when your hopes were high.

When attending any child with pneumonia, it is useful to ask frequently whether sweats and pallor are present. Sweats of course will often be present in a simple pneumonia, but they are often more prominent in empyema and pallor is a rather important sign of empyema. In older children and in adults pallor is perhaps not so pronounced. Still, you have a patient (an adult) who you think is suffering from pneumonia, but the face is not flushed; it makes you feel there is something uncanny about this case of pneumonia. There is in fact some paleness. This should make you think of empyema before unnecessarily blaming the heart. The apex beat often has a tendency to get out slightly beyond the nipple in a patient with empyema. A pneumonia that is not resolving satisfactorily, a case that lags, a patient that looks pale either before an expected crisis or after a crisis or lysis, should make you think of empyema.

The temperature in empyema is irregular. It may be quite normal on the one or two occasions when you happen to "take it" or even more persistently. Empyema may also be a very chronic disease, besides absence of feverishness there may be absence of pain and of dyspnœa and very little cough. But often there is very considerable dyspnœa, particularly in acute cases and one does see empyema without sweats. And some of the patients that later show pallor, are highly coloured earlier in the disease and even livid.

Tenderness evinced on pressure at one spot on a bone may be the only sign of a fracture or fissure. A Colles fracture with the fragments in good position is one of the easiest to overlook and perhaps especially if it is complicating a dislocated wrist which you have just reduced. Most of us find it easy to decide in such a case, but apparently not all of us always. I do not think any of us on finding a tender spot on a clavicle would neglect to do the right thing. Curiously a tibia which was fractured in the lower third puzzled me more than any. The great thing is to err on the proper side.

As illustrating how slight the signs of fracture may be, I mention a little girl who met with an injury that no doctor was consulted about until two years later, despite the fact that the family lived on the lodge doctor's doorstep. There is now a large ugly false joint in the middle of the clavicle, apparently causing no harm except disfigurement.

Vomiting is sometimes seen among the initial symptoms of final heart failure, even in persons who previously have given little or no indication of heart failure and so can be counted as denoting a serious prognosis.

With respect to phimosis in the new-born child, it is readily observable that a foreskin which at birth seems scarcely to require operation, will be relatively longer at six months and worse still at one or two years and this occurs even though the foreskin is dilatable. Practically the only cases that do not take the course that I have mentioned, are those in which without retracting or touching the foreskin you can see the meatus of the urethra and a definite small circle of the glans surrounding it. This amounts practically to a recommendation to circumcise all other male infants; dilatation is unsatisfactory.

A furred tongue in a patient who has had influenza and whose temperature has recently declined, is an indication that the patient is extremely likely to have further trouble if allowed to get up, either complication, relapse or protracted convalescence. But if the patient mentions that

he habitually has a furred tongue, then we need not pay the same regard to it.

"Air-hunger," a term which we typically associate with cases of diabetic coma or impending diabetic coma, is not at all a dyspnœa or rapid respiration—it is the opposite to a rapid respiration. Air hunger is bradypnœa or slow breathing, each inspiration and expiration being carried slowly to a maximum; the result is a succession of long, deep, slow, deliberate breaths and the respiratory rate becomes slower and slower as the condition progresses, even as slow as six per minute they say. Although uræmia does not typically give bradypnœa, yet it may and I have seen typical air hunger in a boy, not yet comatose, suffering from double pyonephrosis; that is, the air hunger was uræmic in origin.

Rapid breathing is a frequent associate of anæmia. Thus a child with leuchæmia presented three remarkable features of which the most noticeable was the extremely rapid respiratory rate, the next was the marble whiteness of the skin and the third was the parotid enlargement on both sides, looking like mumps, as depicted in French's "Differential Diagnosis," page 25.

An intense and recent anæmia in a very sick boy whose illness had begun three years ago with rheumatic endocarditis, was accompanied by a respiratory rate up to 76 per minute. The anæmia caused this very remarkable dyspnœa. In previous attacks of endocarditis he had had such signs of cardiac incompetency as enlarging heart, rapid pulse, œdema of legs and of right side of face and congestion of the lungs. Yet now the heart was not enlarging again, the pulse rate ranged only from 96 to 114, the lungs gave no signs whatever, there was no systemic œdema and incidentally there was no albuminuria. But the breathing rate was up to 76 and the anæmia had become remarkable. This was of course no longer his ordinary attack of rheumatic endocarditis—the signs of mechanical heart failure were not predominant, but the anæmia was, indicating that the valve lesions had now taken on an ulcerative type. (A constant calling out of "don't touch me," even when no one was disturbing him, was an indication of delirium, another sign of ulcerative endocarditis and he later got a paralysis of the left side of face, left leg and left arm due to embolism.)

With further reference to infective endocarditis, the following case may be quoted.

A man took ill with severe headache and feverishness, diagnosed as influenza until the duration of the feverishness sorely taxed all attempts to label it influenza. The headache soon diminished. He had been feeling indisposed for two or three weeks previously, tired and with some headache, so he might have been suffering from enteric fever. The spleen did not become palpable, though its area of dulness was thought to have increased within the first ten days. Red spots on the skin could be found, if one insisted on finding the rose-spots of typhoid. The leucocyte count was 6,400 and a pathologist obtained a response to the Widal test. The nurse who had had experience of enteric fever, insisted that the stools were "typical." Despite all this even at an early stage the diagnosis of enteric fever was recognized as being not perfectly satisfactory. As for the stools, they were merely liquid or jelly-like. The temperature curve was remarkably monotonous: 36·9° C. (98·4° F.) in the morning and possibly 39° C. or thereabouts in the evening with clockwork regularity, exactly the same normal figure every morning and exactly the same high figure every evening. The curve was so even every day that one felt that the condition was not typhoid. With typhoid you would expect some sloping of the curve, either upwards indicating a relapse or downwards indicating improvement. He was not tuberculous, a von Pirquet test yielded no reaction. There was present what seemed to be an old-standing heart murmur. He showed no other signs, never got any embolisms and had no delirium and no anæmia of any severity (at any rate in the earlier stages). Yet he had infective endocarditis and died after a couple of months. The stumbling block to making an early diagnosis was that we seemed to think he ought to have a pulse rate of 120 or more.

What we learned from the case was that the pulse rate in septic endocarditis may be only 90 or 80 and even less and that without the use of digitalis. Secondly, Widal tests were repeated and no reaction was obtained (except in the first one), clearly showing that the condition was not typhoid and that pathological reports may help to mislead. Lastly, as the books mention, there is in septic endocarditis often no increase of leucocytes or very little increase.

In the estimation of the prognosis of heart disease in pregnancy and confinement there are many factors to be considered. It is claimed that an endocarditis of recent origin will give trouble earlier in pregnancy than will a similar old-standing compensated cardiac lesion (probably because the lesion, being recent, is still progressive).

This is exemplified perhaps by a *multipara* who seemed to incur a mitral stenosis a month or two after the birth of a seven months baby and who developed such rapidity of action of the heart (with some further cardiac dilation and dyspnœa) that the only recourse we had was to induce miscarriage after consultation at about three months; the result was very satisfactory.

Old-standing compensated lesions of the cardiac valves, if going to give trouble, will not be likely to do so until mid-term or later, when the indirect pressure from the growing uterus hampers the descent of the diaphragm, displaces the heart and predisposes to stasis in the lungs.

Then as to the actual confinement there are several considerations. To make the confinement easier the child is often small; the size of the uterus at eight months corresponded to the normal size of only about six months in two cases with serious heart lesion attended by me. A *multipara* can expect to fare better than a *primipara,* other things being equal. And finally, a mitral stenosis for very special reasons suffers a special strain.

While she was having an eclamptic fit I was introduced to a *multipara* with an old rheumatic mitral regurgitation and very enlarged heart; she was eight months pregnant, the uterus was the size of a six months pregnancy and the fœtus was living. Several fits occurred; the fœtus perished; confinement followed a month later, corresponding approximately to full time. Heart patients will not tolerate much bearing down, so it is wise sometimes to anæsthetize and save that straining. I was not keen on doing so, as this weakly *multipara* had constant orthopnœa. Her pulse went to 160, but she was delivered unaided of the macerated fœtus. That this happened depended on the fact that she was a *multipara* and that the uterus and the fœtus were small. I have not many notes comparing her condition before and after labour, but after labour the liver was down to the umbilicus.

Obviously the stress and fatigue of a confinement play havoc with a heart that is already failing.

A *primipara* told me that she had once spent six months in a sanatorium out west because hæmorrhage from the lungs was supposed to be due to tuberculosis and she was then discharged because there was no consumption and the hæmorrhage was found to be due to heart disease. Mitral stenosis was her trouble. At the seventh month of pregnancy the legs became very swollen, but there was no albuminuria. At the eighth month there was albuminuria, a very considerable quantity. The lips got blue at times. The left hand swelled. The legs were extremely œdematous and also the vulva. Four Southey's tubes were put in each leg. Labour began just before full time. After some hours in the first stage of labour the external jugular veins in each side of the neck were pulsating greatly, indicating the overloaded condition of the right side of the heart. I had no assistance except that of a registered midwife who had asked me did I expect any trouble. I explained to her that when the uterus contracts and retracts during labour, it forces blood from the veins of the large uterus onwards into the right side of the heart, which then sends the blood through the lungs and into the left side of the heart. But the left side of the heart is partly blocked by mitral stenosis, so that an increasing quantity of blood gets dammed back into the right side of the heart. This is the special difficulty in mitral stenosis; other forms of valvular trouble will at any rate give the blood a chance to get through. I must save the patient the strain of bearing down and must apply the forceps immediately on or soon after the completion of the first stage. But whether such a patient delivers herself or is delivered instrumentally, a time of greatest risk is just when the child has been born, because at that time a large quantity of blood is driven out from the contracting uterus, first into the veins and thence is carried to the right side of the heart.

The first stage having been completed, I soon induced anæsthesia with chloroform, put a tourniquet loosely on the arm and applied the forceps. The nurse withheld the chloroform for some time before the child was born. When the child was born, the patient suddenly got very blue. I immediately opened a vein in front of the elbow; it bled very freely. The patient's pulse could not be felt at all at either wrist, the breathing consisted of a very occasional short gasp, the eyes were filmy, staring and fixed and the cornea reflex was not responsive. The nurse had been instructed not to mind the uterus; we wanted it also to bleed and fortunately it did. The patient recovered temporarily; her chest was now full of râles, corresponding to back pressure in the lungs. Having secured the afterbirth we propped the patient up in bed and I left her very pale, very tired, considerably cyanosed and with the apex beat quite five centimetres (two inches) external to the left nipple, whereas before confinement the apex beat had been internal to the nipple. Of course the apex beat was formed by the right side of the heart.

The child weighed 2·4 kilograms (five and a half pounds) and survived.

Within twenty-four hours the mother's apex beat came back within the nipple line; the pulse rate was 124, the respiration 30. A week later moist sounds were abundant in the left lung posteriorly and breath sounds could scarcely be heard at the base of the left lung. The liver was five centimetres below the costal margin. Epigastric pulsation (a sign of enlargement of the right side of the heart) was very considerable. The apex beat was in the sixth space. Dulness was present well out in the second and third left interspaces, indicating no doubt an enlargement of the left auricle so often seen in mitral stenosis and as the patient began to get hoarse, I judged that the left auricle was pressing on the recurrent laryngeal nerve. This also is well known to occur in mitral stenosis. The patient died seventeen days after the confinement.

Another woman that I have twice confined and whom I consider to be perfectly healthy and free from heart trouble, once gave me anxiety. The child had just been born when the mother seemed suddenly to get very "chesty." The chest was now suddenly full of râles, large and small. She had had a little chloroform. In less than fifteen minutes she was quite right again without blood letting.

This case can be compared with the other. It is not necessary to suppose that the patient had a mitral stenosis, though the signs of mitral stenosis can be very obscure; but the blood from the uterus, thrown into the right side of the heart, was not carried sufficiently quickly through the left side of the heart. The condition could be called acute œdema of the lungs; I have read that it may occur at childbirth, but I see no reason to imagine what also I have read, that it should be looked upon as being reflex.

POINTS FROM PRACTICE: TREATMENT BY DRUGS.[1]

By F. G. MEADE, M.D., B.S. (Melbourne),
Brisbane.

WHEN the matter of saying a few things about treatment by drugs was brought before me by the Secretary some little time ago, the first thought that came to me was this: that I knew very little about drugs, that I did not really believe in them much and felt sure that anything I could say would not be of much importance or interest to members.

Though a little further reflection helps me to modify this somewhat and inclines me to realize that drugs—some drugs—have really some virtue, yet it is still difficult to assemble ideas that would prove worthy of being brought under your notice.

In the first place it may be stated that whilst the use that is made of drugs is very considerable, prescribing being one of the chief occupations of a medical practitioner, still the business of doing so is not as a rule one that is enthusiastically engaged in or one that seems to have much interest for the average practitioner.

Indeed drugs, like Cinderella, are depended upon and are expected to do a very great deal in the way of curing our patients and of making our reputations; but I am afraid they, like her, are never held in great esteem by the medical fraternity generally.

This reputation is hardly a just one, for if we think for a moment what a serious loss we should sustain if we were suddenly bereft of all drugs now at our command, we must realize that we should be at once badly off. How, for instance, would we relieve pain if we had no morphine or other analgesic; what would we do if we had no cocaine or its substitutes for our minor surgery, no chloroform or ether for our anæsthesia and no antiseptics?

As, judging by the title of these papers, I am to concern myself with points from practice, an effort will be made to follow along these lines.

Drugs are used rationally or empirically. The former method, implying a more or less complete physiological knowledge and also exact ideas about the pharmacology of our remedies, would seem to be the more desirable to practise. But it is seldom that we have the requisite knowledge, so that almost

[1] Read at a meeting of the Queensland Branch of the British Medical Association on May 7, 1926.

always we have to fall back on to empirical methods, making use of our experience of the action of drugs as we can learn them in the consulting room or at the bedside. I shall therefore disregard the former method and confine my remarks to some examples of the empirical use of drugs.

There are many conditions in which pain is the most prominent feature and to which the term fibrositis is frequently applied and of which an example is lumbago—often a long and tedious complaint. When the diagnosis is clearly established, what can be done to relieve? If the patient is seen early, it would appear wise to give effective doses of opium or a derivative suitably corrected to prevent constipation. Milder analgesics are not so dependable. The important point is to get in early and repeat the dose as often as necessary and thus keep down the pain. A case may in this way be cut short.

Similarly in cases of renal and biliary colic morphine given early seems to act more quickly than when the pain has been allowed to persist for some time. It has been noticed also that unless the dose given is an adequate one, the second requires to be almost as large, so that the total amount required is greater than a single adequate dose.

There are also cases of irritable dry cough that follow influenza and similar catarrhal conditions involving the trachea, especially, it seems, the subglottic areas. Many of these coughs are distressing and persistent. They are difficult to control with the ordinary cough mixtures. It has been found, however, that effective doses of morphine, preferably combined with an acid, will usually do so and save the patient a lot of discomfort. These coughs, unless thus treated, often last for weeks. The explanation of all these cases seems to be the same. Apparently the continuous pain impulse, like the repeated cough impulse, gives rise to an irritable focus in the cord which "facilitates" the production of the main symptom and thus keeps up the morbid condition, whereas the morphine would seem to hamper the passage of the peripheral stimuli and thus raise the synaptic resistance in the cord. A thought that often comes to mind in regard to this troublesome cough, is that "the cough aggravates and keeps up the cough."

Of course there is a natural disinclination to make use of opium and morphine and one endeavours to avoid doing so if possible. As a substitute or partial substitute in case of pain it has been found that an ordinary dose, say, 0·6 gramme (ten grains), of acetosalicylic acid together with a small dose, say, 0·6 mil (ten minims), of *liquor morphinæ hydrochloridi*, equivalent to 0·006 gramme (one-tenth of a grain) of morphine hydrochloride, will, if given by mouth, have almost as much effect in relieving pain as 0·015 gramme of morphine given hypodermically. It is a combination that is much more effective than would be expected.

Incidentally I may mention a type of patient to whom morphine should be given only after most careful consideration. He is the large obese person, about fifty years of age, who is often a potential if not an actual diabetic. Such a person may slip into a coma very quickly and be found very difficult to arouse. Perhaps a carefully determined dose of "Insulin" would help to avoid a catastrophe.

About calculi, salivary calculus is not often seen.

A man about thirty years of age had noticed a swelling below his left jaw at meal time for a few weeks. Examination revealed an elongated stone in Wharton's duct. Surgical treatment seemed to be indicated. However remembering what should never be forgotten that a surgeon is a physician first, I gave him a mixture containing potassium citrate and iodide. This was given an hour before meal time with a large draught of water.

It is well known that potassium iodide is very quickly excreted into the mouth apparently through the various mucous and salivary ducts. This line of treatment was based on this fact, whilst some dependence was based also on the idea generally held that the citrates increase the fluidity of the blood plasma *et cetera*. The sequel was that the patient brought along the calculus in a matchbox for inspection in a few days time.

Calculi are much more common in the urinary tract. They seem to be particularly prone to form in men who work hard and sweat much. Frequently the diagnosis is not easy, though pain referred to the renal and ureteral areas should lead one to a conclusion. The treatment, it should be carefully remembered, is not always surgical by any means. Large doses of potassium citrate, say, four grammes (one drachm), with copious draughts of hot water together with a protein free diet to keep the urine as alkaline as possible will very often succeed in bringing about the passage of the stone *per vias naturales*. This treatment may not succeed at once and may require to be persisted in for a time and this should always be done. It is my belief that the greater number of ureteral calculi at any rate do pass in time if encouraged and given an opportunity to do so.

A few weeks ago an elderly woman was attended for a partial intestinal obstruction, characterized by an intermittent abdominal distension and spasmodic abdominal pain. She had been treated eighteen months previously for gall stones. On this last occasion paraffin was given regularly; large enemata of water were used and she was kept on a soft, easily digestible diet. She improved slightly in the course of a few days. She was then given a mixture containing sodium bicarbonate and tincture of belladonna in 0·6 mil (ten minim) doses every six hours. Next day she passed a cylindrical stercolith, very hard and heavy, and about 6·5 centimetres (one and a half inches) long by five centimetres in diameter. Great relief followed.

The explanation tentatively offered is that a gall stone formed the nucleus of the stercolith; that this eventually found its way down into the lower ileum; that the belladonna relaxed the musculature of the ileal wall and the ileo-colic sphincter, thus enabling the stone to pass on into the colon and to be evacuated.

Similarly belladonna appears to be of use in lessening the tendency to pyloric spasm and combined with morphine gives more and quicker relief in those attacks of pain called biliary colic than morphine by itself. In these cases of so-called biliary colic it may be suspected that there is often a larger element of pyloric spasm than there is usually supposed to be.

Stramonium is a member of the belladonna group and in asthma a combination of the tincture in about 0·48 mil (eight minim) doses together with about 0·36 gramme (six grains) of potassium iodide and a routine dose of paregoric seems perhaps better than anything else, with the exception of adrenalin given hypodermically, to relieve and lessen the paroxysm. It may be regarded almost as a specific. It is very seldom found to fail. Another very important point in the treatment of asthmatics whose lungs are often emphysematous, is to keep down the infradiaphragmatic tension to allow easier lung action.

In speaking of aperients one is not quite sure that the use of paraffin which was recommended years ago by Lane, is quite thoroughly understood especially by the younger members of the profession. It must be borne in mind constantly and it must be dinned into patients that paraffin itself is simply a laxative and lubricant and is not an aperient. A bowel that is inclined to act may not need any more than the paraffin to insure evacuation. Generally, however, an aperient is required and should not be omitted. The rule is to give paraffin of the proper viscosity, in say fifteen mils (half ounce) doses, regularly the last thing at night and the first thing in the morning and every night or at least every other night a suitable dose of, say, cascara. The liquid extract of cascara is preferable, as the dose can be more carefully determined and there is almost certain action which is more than one can be sure of when pills and tablets are used. Moreover, in prescribing cascara it is absolutely essential in order to secure results that some well known brand be used. The cascaras of commerce vary much in potency.

Concerning drugs in the treatment of gastro-enteritis or enterocolitis, the treatment of this condition in children is one of great importance and it behoves every medical practitioner whose duty it is to treat these patients, to learn as much as possible in regard to them. Perhaps there is no class of case in which management counts for more. In these cases there is scope for treatment based on physiological and pathological knowledge. *Oleum ricini* should be in the locker of every medical practitioner who treats children. But more important still he should know how to use it, when to use it and when not to use it. Undoubtedly it is often abused by all concerned. In children it seems that the first requisite is to secure a complete emptying of the whole bowel to get rid of all food stuff. A good dose of castor oil will almost certainly do this. Repeat the dose at once if it be vomited, for the second dose usually keeps down. Having cleared the bowel, give no more oil. The use of repeated doses is not easy to understand. Once the bowel has been emptied, keep it empty of all food likely to leave a residue that might irritate the mucosa or provide pabulum for organisms—that is, all intake must be fluid and absorbable in the upper part of the food tube and it must be fed from a spoon in small quantity at a time. Provided this is done, no castor oil or other aperient will be required. A wash out, say of normal saline solution, once or twice daily will look after the lower bowel; the upper part of the bowel will look after itself. This will be understood if the physiological fact be remembered that the contents of the bowel are fluid as far as the hepatic flexure at least. It is really only in the descending colon that any stagnation is liable to occur. The recognition of this is important particularly during the recovery stages, because the injudicious use of aperients may relight up the whole trouble. If after the bowel has been cleared of all food, bowel action is still frequent and is exhausting the child, small doses of laudanum may be required. This should never be prescribed with other ingredients in a mixture, but always in dilution alone so that it can be used just when required and ceased at once as soon as the indication for its use has come to an end.

Acidosis in these cases is liable to occur very soon for well known reasons. The administration of sodium bicarbonate and sodium citrate in doses of a few grains every two or three hours and given in combination with a carminitive is advisable. The giving of soluble carbohydrate at the same time in the form of cane sugar, barley sugar or boiled sweets is practised. Whey, barley water and rice water suitably flavoured must be depended on for some time until the bowel action moderates and the stools are becoming formed and of brown colour.

Bismuth is not a drug to be depended upon much, especially in the early stages. In the later stages if an astringent seems desirable, it can be used. The treatment of these conditions has been mentioned at some length principally to indicate the inadvisability of depending much on drugs. Most patients will get better if treated along these lines.

Perhaps it would be permissible to refer to the use of drugs generally. The standard of the medical profession in regard to drugs is the British Pharmacopœia. Anything not contained in that volume or in Squire's "Companion" will almost certainly be found in Martindale and Westcott's "Extra Pharmacopœia," a veritable mine of medical information which no medical practitioner can afford to be without.

Anyone, however, who has done dispensing work for any length of time, cannot but be surprised at the way in which some prescribers almost entirely neglect simple pharmacopœial drugs and order in their stead proprietary lines, often with high sounding names, the products of enterprising manufacturing firms. Of the ingredients of these preparations it cannot infrequently happen that the prescriber has but the faintest knowledge and he depends almost entirely upon the manufacturer's statements as regard the indications for their use. Another drawback is that they are usually expensive. Their saving grace is palatability, but every medical practitioner should have sufficient pharmaceutical knowledge to be able to prescribe pharmacopœial drugs in palatable form. The use of these more or less patent medicines no doubt is often

attended by success, but if that success can be achieved by the use of the simpler drugs, it would seem more desirable to use them. The use of proprietary lines undoubtedly tends to lead to self-medication, a habit which is well seen in connexion with a common analgesic.

To supply a want of medical practitioners in this connexion there is published a small book called the "Australian Pharmaceutical Formulary," briefly styled the "A.P.F.," which contains a lot of formulæ yielding excellent preparations which can be used as substitutes for these expensive and widely advertised imported proprietary preparations. Pharmacists in general stock these "A.P.F." preparations and usually show a desire to encourage their use; so far as I am aware they have no other interest than the promotion of better prescribing practice.

There is great need also for prescribers to refrain from haphazard methods in regard to the use of even simple drugs such as borax and soda and salt. One not infrequently hears for instance that a patient has been told to make a nose wash by mixing so much borax and salt *et cetera*. Now there is need for exactness even here. The lotion when ready for use should usually be isotonic with the blood plasma *et cetera*. Is that achieved by these rough and ready methods?

Experience teaches the need also of being careful in giving directions to patients as to the times of their taking medicine. For example the common direction "three times a day" may mean several things; it may mean every three, four, six or eight hours or it may mean in practice to be any irregular interval of time so long as three doses in the day are taken.

It may be necessary to take the drugs in some relation to meals. If so, the patient can be instructed one hour or half an hour before or after meals as the occasion requires.

Let us, however, take such a drug as digitalis being given, say, in a case of auricular fibrillation. At first it will need to be given at frequent intervals, say every four hours day and night, if possible. After the irregularity has been controlled and the pulse rate has been brought to a safer level, it will ally in the case of people who rise late and go intervals. What is wanted is a regular dose right round the clock, say three times in the twenty-four hours or every eight hours. This will be secured by giving it at 6 a.m., 2 p.m. and 10 p.m.; occasionally in the case of people who rise late and go to bed early, one will have to compromise and give it, say, at 7 a.m., 2 p.m. and 9 p.m., every seven hours. The important point however is to cut out slip-shod methods.

Incidentally it may be mentioned in connexion with digitalis medication that there has recently been put on the market by Messrs. Parke, Davis & Company a new digitalis tincture No. 111 or "Digifortis." It is a carefully produced drug and has proved very satisfactory in use.

POINTS FROM PRACTICE: OBSTETRICAL.[1]

By CLIF. TUCKER, M.B., B.S. (Melbourne),
Brisbane.

WHILE I may not hope to tread any new paths tonight, yet the recounting of some of the difficulties and perplexities which continually meet the obstetrician, may not be without stimulus.

The first query we have to answer is: "Am I pregnant and, if so, when will I be confined?" These questions, in the first two months of an amenorrhœa, are sometimes very difficult to answer.

Conception does most commonly occur in the first week following the cessation of the menstrual flow, but by no means always and when we remember that menstruation can and not infrequently does occur until the products of conception fill the uterine cavity, absolute reliance cannot be placed on this relation in estimating the actual date of labour.

Personally I have not found accuracy possible in estimating the date of confinement. Possibly in these warmer climates the act of conception does not bear the same close relation to the cessation of the menstrual flow as in colder climes or perhaps the class of patients I have had to deal with, are unusually careless in the recollection of their menstrual dates.

A reckoning of two hundred and eighty-one days from the first day of the last menstrual period, as is done in Burroughs and Wellcome's diary, gives too short a period. I add six days on to the date in Burroughs and Wellcome's tables and then tell the patient it may be a week or longer one way or the other. Tweedy reckons seven days ahead of the last day of last menstrual period and then calculates three calendar months back, this gives about the same date as the previous method.

The classical signs and symptoms at the early months of pregnancy and the diagnosis of the condition, I will pass over except to say that if in the first two months you are unable to be certain of pregnancy, you will not find it damages your standing with your patient to explain matters and tell her candidly that in one or two months you will be able to tell her definitely, but that at present you cannot do so. At this stage a detailed examination of your patient, especially of the mouth, the recommendation of the removal of all carious teeth and the cleaning up of any pyorrhœal condition is advisable. For this I recommend a saturated aqueous solution of thymol and if there is not considerable improvement in two weeks, the removal of such teeth as the dentist recommends.

Of those abnormalities which are more commonly met with in early pregnancy, the vomiting of pregnancy rarely becomes worse than an annoyance. The regulation of diet and exercise, the rectification of any gross abnormalitity such as a retroverted gravid uterus by the use, if necessary, of a

[1] Read at a meeting of the Queensland Branch of the British Medical Association on May 7, 1926.

rubber pessary to prevent a return of the malposition and the administering of extract of *corpus luteum* are generally efficacious in checking it.

I have used Oppenheimer's "palatinoids" and have had sufficiently encouraging results to rely more on them than on any other therapeutic aid. Bismuth I have found uncertain in its action as one would expect; its tendency to constipation is also an objection.

Perhaps the next most common complication at this stage is a sudden loss of blood of greater or less amount *per vaginam*. The patient will generally give some history of a fall or heavy lift preceding the flood; this of course may or may not have had anything to do with the causation.

The flow may have been scanty or severe, indicating a threatened termination of pregnancy. It may be possible, when the patient is first seen, to determine whether the abortion is inevitable and when the products of conception are exhibited if complete or incomplete.

In all cases in which the condition is only threatened, rest in bed and the administration of a sedative mixture containing bromide of potash, 0·6 gramme (ten grains), liquid extract of ergot, 0·6 mil (ten minims) and tincture of hyoscyamus, 0·6 mil, a light diet and the withholding of severe purgatives seem the limit of activity. My practice in these cases is never to make a vaginal examination, if there is a chance of preventing the abortion; this chance will not be enhanced by any manipulations of the uterus or cervix. If the abortion is inevitable, it is unnecessary and there is the possibility of the introduction of sepsis. Once the abortion becomes inevitable as evidenced by repeated hæmorrhages, passage of large blood clots, intermittent pains and the expulsion of whole or part of the products of conception and unless the whole of the fœtus, membranes and placenta can be demonstrated, I advocate the following procedure in all cases, namely, assume that the abortion is incomplete, curette and flush out the uterine cavity. At the time the patient is first seen the vagina is tightly packed with iodoform gauze; this controls the hæmorrhage and can be left in for twelve hours if necessary; at the end of this time the cervix will be dilated, the uterine contraction will have been stimulated and placenta will perhaps be found lying on the gauze. It also gives one time to get the patient to a hospital and choose a convenient time for your operative measures. The hand is not allowed to come into contact with anything that enters the vagina or uterus and the curette is done with a broad ended blunt instrument gently and thoroughly. With care this can be done without danger to the uterine wall and one can be quite certain the cavity is empty; it is then flushed with an antiseptic lotion. The late J. B. Murphy, of Chicago, in 1914 advocated the procedure in the following terms:

> When the patient has an impending miscarriage without other pelvic conditions which force you to make an examination, no examination of the condition of that vagina or uterus must be made.
>
> Never put your hand across that threshold, because you will carry into that vagina saprophytes. Decomposition takes place and then following miscarriage comes infection. Invariably the infection of a miscarriage has its source from without. If the miscarriage is going to take place, your examination will do no good. If it is not going to completion, your examination can do harm. When you discover some of the products are retained do not stop until the uterus is cleaned out and completely cleaned out.

With the impossibility of knowing what infection has been introduced before we come on the scene and the sinister reputation these patients enjoy of becoming septic, a sense of personal security is enjoyed in knowing that at least one avenue of infection has been kept closed. At the seventh month and a half the patient should be examined thoroughly abdominally and *per vaginam*, abdominally for the lie of the child and for any abdominal complication or abnormality, vaginally to ascertain any pelvic abnormality and to estimate if possible any likely disproportion between the size and shape of the pelvis and an average sized head. At this stage one will be able to detect those gross pelvic abnormalities which absolutely preclude the passage of the child. It will not be possible in those very much more numerous borderline cases in which we have a relatively diminished or abnormal pelvic inlet or outlet, to say what are the chances of a birth *per vias naturales*. It is in these cases that there is only one sure way of determining it and that is by a test of labour completely carried out, even if we end up with a dead or lacerated child, we have this definite information for our next pregnancy and before we take the grave responsibility of performing a Cæsarean section, grave not because of its immediate attendant risks so much as the condition of that uterus. To a woman in the early child bearing period such a uterus at least potentially weak is a sword hanging over her head.

When we consider the numerous cases which we have felt certain could not terminate naturally when the head has ridden on a relatively contracted pelvic brim at the onset of labour and a stormy time seemed inevitable and how finally with comparatively little trouble a birth has occurred, it makes us increasingly cautious of advising a Cæsarean section in a *primipara* or one who has not had this test of labour.

We are dealing here with a very variable factor, the fœtal head, which in the same weight babies may be large or small, capable of big variations of shape from very little alteration to extreme moulding in its passage through the canal. In these cases a waiting policy, if irksome and tedious, will be in the best interests of mother and child.

The monthly examination of the urine to the seventh month and a half and then bimonthly and weekly in the last month is advisable to guard against eclampsia. The rapidity with which a clear urine can become "solid" with albumin is astounding. Fortunately these patients if got early generally respond to treatment. I have found absolute

starvation most effective, carried out in hospital. Nothing but water is given and sufficient purgative castor oil or magnesium sulphate is given to insure at least eight water motions a day. This can be carried on for a long time, in a recent case for sixteen days, when fortunately labour commenced and a healthy child was born.

Of the actual handling of a patient in labour I only wish to stress the point that the actual delivery of a woman is essentially a surgical operation, demanding the same aseptic routine as for our other surgical procedures.

Here unfortunately in many of the maternity homes and in the patients' own homes this ideal seems impossible of attainment. A Boothville, the Salvation Army Maternity Home for single girls, the routine followed is to shave the pubic hairs, clean the parts with lysol solution and just before delivery to paint the vulva, perineum and inner sides of the thighs with 1:1,000 "Acriflavine." No vaginal examination is made except with the gloved hand. I have trained them to put on the glove without touching the outside with the ungloved hand.

In approaching a patient in labour as in every other surgical or medical problem, the crux of the position is your diagnosis. (i.) What is the position of the child in relation to the maternal pelvis? (ii.) Can the child be born naturally? This latter is more particularly an antenatal problem as indeed is the first.

Much valuable and generally all necessary information can be obtained by an examination of the abdomen. By inspection there can be noted the regularly rounded contour of the abdomen from the *symphysis pubis* to ensiform outlining the fœtal back or shoulders as contrasted with the more flattened abdomen with the irregular outline of the limbs denoting a posterior lie of the fœtus. The palpation of the fœtus through the abdominal and uterine walls is not always easy, but no effort should be lost of informing ourselves of the fœtal lie by this valuable method in between the pains and if necessary under an anæsthetic. The fœtal parts can generally be accurately mapped out and the question of a vertex or breech presentation determined and a normal anterior or posterior vertex presentation diagnosed. Also, if the head is engaged in the pelvis, we know that no obstruction will be offered to the passage of the fœtus by the pelvic inlet.

In the presence of much fat or *liquor amnii* recourse may have to be made to vaginal examination to determine the lie.

After the patient is anæsthetized, if an examination with two fingers of the presenting part distorted with a *caput* leaves one in doubt, the whole gloved hand can be insinuated into the vagina and by a digital examination of an ear the absolute position becomes evident. The relative proportions of fœtal head and pelvic inlet and outlet, the chances of an unassisted birth or the necessary artificial aid required can be fairly accurately gauged by this means.

At this stage it is a great help to pause and visualize the fœtus just as it is lying in its normal or abnormal position, especially is this so in a posterior lie or an intermediate one, when if the cervix is sufficiently dilated the posterior can be converted to an anterior position with the aid of the other hand on the abdomen turning the body with the help of an assistant on the other side of the body. This can be generally effected with the head in the hand or the hand can be slipped past the head and a shoulder turned with the fingers.

It will enable one to deal with that most troublesome of complications, the prolapsed cord. After many painful experiences of this condition with endeavours to replace the cord by hand, mechanical means or by posture, I have come to the conclusion that the safest procedure for the child is to convert a vertex to a breech presentation, when the cord will not be severely compressed except by the after coming head and then instead of a loop of cord one thickness only is compressed and the danger is no greater than in an ordinary breech.

Waron estimates the fœtal mortality at as high as 10% in *primiparæ* which seems to me to be a higher figure than experience would lead one to expect. In the management of a breech case I think it important, once the hips are born, to wrap the fœtal body in a warm towel and leave it alone, concentrating on assisting the abdominal muscles and uterine pains by pressing on the fundus, getting well up over your patient, kneeling on the bed if necessary and helping to push the fœtal shoulders and head through the pelvis. By this means the fœtal head can be kept well flexed going through the pelvis and there is less liability for the arms to extend or for damage to the fœtal skull and meninges to occur. The cause of the fœtal mortality in breech cases has been found at *post mortem* examination to be due to a tearing of the blood sinuses at the base of the skull with resultant hæmorrhages and with the head fixed in the pelvis traction on the body tends to separate and tear the basal meninges. Pituitrin I use in every case in one cubic centimetre dose, if it is required before the birth to assist the uterine pains, this of course within strict and definite limits or in 0·5 cubic centimetre dose after the birth of the child as a help in the contraction and retraction of the uterine muscle and the expulsion of the *secundes* and to minimize the risk of *post partum* hæmorrhage.

I will not consider the difficulties or indications for the use of forceps, but certainly owing to the demands on our time and the conditions under which we have to work, our obstetrical work is frequently hurried and forceps are used oftener and earlier than desirable. It is inevitable that we should have pondered rather sadly the report of the Royal Commission on Health in the Maternity Hygiene Section:

Since the advent of the maternity allowance the number of the mothers who were attended by medical practitioners

at their confinements increased from 63·2% to 79·3% and the maternal mortality increased from 5·1 to 5·4 per 1,000 births.

In 1923 of total female deaths in the childbearing period, one-fifth were due to tuberculosis, one-eleventh to cancer and one-eighth to childbirth, in a process that should be a normal physiological one; this does not take any account of morbidity or invalidity.

The report states that all witnesses were agreed that the results of obstetric practice had not improved proportionately with advances in medicine and surgery.

We must ask ourselves why.

Of the deaths roughly one-third were due to sepsis, one-third to the toxæmias of pregnancy and one-third to other complications. There should be a diminution from all these causes, especially the two latter by systematic antenatal care, that most valuable branch of preventive medicine.

In Queensland 61% of patients in 1924 were treated in hospital, though I should say a smaller percentage than this in the city. I am afraid that many of the private obstetric homes are no better or even worse from the hygienic standpoint than the patients' own homes. The figures of Queen Victoria Hospital, Melbourne, show the great value of antenatal care in lessening morbidity and mortality. In three years there were 1,031 confinement cases with two deaths. In 1924 429 cases, no deaths. No eclampsia cases occurred and no grossly syphilitic infants were born.

The Commission's suggested remedies to improve this state of affairs is worthy of our close study and assistance to attain them.

They are as follows:

The most important measures that should be undertaken for improving maternal mortality and morbidity is the adequate provision in populous centres of properly equipped and efficiently staffed maternity hospitals either public or intermediate. These would not only give better accommodation for maternity cases, but would afford the much needed facilities for training obstetric nurses and medical students. Prenatal and post-natal clinics as well as observation wards should be associated with these hospitals.

Their recommendations were that:

1. A division of maternity hygiene be established in the Commonwealth Department of Health.

2. Conditional subsidies be granted to States to provide facilities for attention to women before, during and after childbirth according to standards approved by the department.

3. Conditional subsidies be granted by the Commonwealth to assist the education of medical students and nurses in obstetrics.

4. *Maternity Allowance Act* be amended: (i.) to insure prenatal supervision, (ii.) to define a viable child as one measuring at least thirty-five centimetres (fourteen inches) in length.

These recommendations give one hope that in the near future financial aid will be forthcoming to establish and extend the maternity hospitals and that the expectant mother will at least be relieved of all financial worry connected with her parturition.

I am not certain that the present arrangement is a good one, nor convinced that the general practitioner who at present does the bulk of the obstetric work, is the best person to conduct the confinement. Much of his obstetric work when conducted in ill equipped homes at a distance from his surgery must be hurried and hence ill done. Considerations other than the needs of mother and child have frequently to determine the use of forceps, another obstetric case three or four miles away demanding immediate attention; the hundred and one calls on a busy practitioner's time make it impossible for him to sit back and allow things to take a natural if slow course which is so essential to good obstetrics.

The *Midwife's Act* in England limits the obstetric work a midwife may do, to those cases in which no abnormality or complication exists. Should such exist, a medical practitioner must be consulted.

Normal pregnancies, labours and puerperia are physiological processes and a well trained obstetric nurse can attend and manage these cases and in a hospital or institution she has the time to give to them which a general practitioner cannot.

With careful antenatal care and a weeding out of cases other than normal by a competent medical man more of these patients can be attended by trained nurses in hospitals with benefit to the mothers; this is the practice followed at Boothville, the Salvation Army Maternity Home, where the patients are single girls; here it is found that approximately 90% of the girls are attended by the nurses alone, in about 10% a medical practitioner is called in for the confinement and in another 10% of cases after the birth for torn perinea or some complications, such as retained placenta after birth. If we had an extension of the formation of intermediate hospitals large enough to utilize the services of a trained nursing staff who could conduct all the normal and uncomplicated confinements with one or more resident obstetricians with ante- and post-natal departments attached, a substantial advance would be made.

Medical and nursing education to be effective, must be progressive, continuous and coexistent. The nurses' education will not be complete and comprehensive as it should be until we have a medical school attached to our University where facilities for graduate and post-graduate education will exist to equip us for our obligations to the public and as teachers to the nursing profession.

In all parts of the Commonwealth the members of the profession are stirring themselves to keep in the van of the progressive movement of preventive medicine and in no branch of the science will such satisfactory results accrue to the general community as in obstetric work, but the work and our education must be continuous and progressive and always the greatest stimulus comes from the medical school or its offshoot.

LEPROSY ACQUIRED IN A LAZARET.

By GEORGE R. HAMILTON, M.B., Ch.M. (Sydney),
Honorary Assistant Dermatologist, Sydney Hospital; Honorary Dermatologist, Royal North Shore Hospital; Honorary Dermatologist, Royal South Sydney Hospital, Sydney.

CONTACT with lepers has been regarded as a frequent cause of leprosy. In regard to the case herein reported certain special features should be noted:

(i.) The patient, a white man, was an attendant at a lazaret.

(ii.) He was in close association with leper patients.

(iii.) Whilst at the lazaret he accidentally cut his left hand.

(iv.) Thirteen years later he manifested signs of leprosy.

(v.) The first sign of the disease appeared close to the scar of the cut.

(vi.) He has not been in any other countries but Scotland and Australia with the exception of a trip of one week's duration to Iceland in company with the late Jonathan Hutchinson. In Australia he has been in South Australia, Victoria and New South Wales and has not travelled north of Brisbane in Queensland.

(vii.) He has an undoubted right to compensation under the Queensland *Workers' Compensation Act*, 1916.

The importance of these points may be emphasized by reference to the literature on the subject. Many means of infection have been postulated in leprosy, such agents as dust, water, fish, blood sucking insects *et cetera* being incriminated, but nothing definite has been proved in regard to any of them. Experiments with inoculation into animals have been only partly successful. Among the recorded cases in which inoculation in human beings has been performed, that of Arning is prominent. In this instance a Sandwich Island criminal (Keanu) was inoculated with a leprous nodule.

Two and a half years later Keanu developed all the signs of leprosy, but in his case there was a family history of leprosy. A report made by Hatch, of Bombay, and quoted by Crocker is rather interesting when compared with the way in which my patient became infected. Hatch's patient was a student who cut himself whilst making a *post mortem* examination on a leper; this was followed by symptoms of leprosy. Macleod also reports a medical man who confined a leper negress and while doing so wounded his finger; it was noticed that the wound took a long time to heal. The doctor eventually became a typical maculo-anæsthetic leper. That nurses and attendants on lepers have been known to contract leprosy is also reported by Macleod. Moreover, Crocker says:

> One fact stands out clearly, that intimate association with lepers is fraught with danger. Even in India the non-contagionist commission showed that 5% of those who live intimately with lepers contract the disease.

That the disease can in some way be spread from human being to human being is well shown by Benson's case of an Irishman (Hawtrey), who acquired the disease in the West Indies and went back to Ireland. While he was there his brother used to sleep with him and after his death wore his clothes. Five years later the brother manifested all the signs of tubercular leprosy. There is also the famous case of Father Damien de Venster who was in residence with lepers at Molokai in 1873 and who developed leprosy in 1882. Again, it is interesting to note that in certain islands, New Caledonia, Mauritus and Rodriguez, where, according to Castellani, leprosy was not previously known, the infection spread, it is believed, from the introduction of a single leper.

Macleod has drawn attention only this year to the contact cases of leprosy in the British Isles. He records four infections due to contact in order to correct "the impression and to show the danger of belittling the contagiosity of the disease among certain members of the medical profession and laymen, that leprosy in the British Isles is either non-contagious or its contagiosity is so slight as to be negligible." Further he records the resolutions of the second Leprosy Congress at Bergen in 1909, one of which is:

> That leprosy is a disease that is contagious from man to man, whatever may be the method by which this contagion is effected.

Clinical History.

G.N.T., aged fifty-six years, single, was born in the Isle of Bute, Scotland; he is an analytical chemist by profession. In 1897 he left Scotland in the "windjammer" *Loch Lomond*. No port of call was made between Greenock and Melbourne, where he disembarked and took up duties with a firm as an analytical chemist for two years. He then went to Port Pirie and Adelaide and back to Melbourne for various periods. In 1904 he went to Brisbane to the Department of Agriculture and later to the Board of Water works and to another firm. All this time he worked as a chemist. He lost all his positions on account of alcoholism. Being out of work and "down and out" he went to Peel Island in October, 1912, as an attendant in the white section of the lazaret. According to him there were at that time about fifty patients manifesting all types of leprosy. Six weeks after being at the lazaret he went to Beach Camp in the lazaret. There the cook in the blacks' camp was endeavouring to cut off the end of a piece of wood with an ax. He took the ax from the cook and, placing the left hand on the wood to steady it and using the ax with the right hand, was actually striking when the cook moved behind him and distracted his attention; the result was that he cut the ring and middle fingers of his left hand. He remembers distinctly that the cut took some time to heal, about three weeks He was at the lazaret for four and a half years. His duties were to see that the patients were fed, to keep the place clean; this included scrubbing; to attend on bedridden patients, washing them and often doing dressings in the absence of dresser or nurse. He never wore gloves. He did no dressings while he had the wound on his left hand. Making beds while patients were in them, were among his duties. One blind leper in particular got most attention. Some of the patients had open sores; of the white patients two had open sores, on fingers in one case and in the other on the neck. There may have been open sores in the black patients, but he did not see much of them. He slept in quarters about a hundred yards away from the patients' huts. No leper was ever in his quarters. He always washed his own clothes. Lepers never handled his clothes or food, but he used to take his meals and have

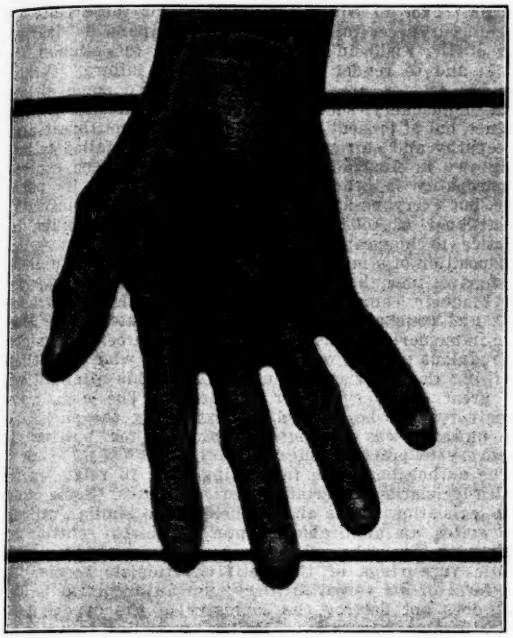

FIGURE I.
Showing Lesion on Back of Patient's Left Hand. The scar from the ax wound may be seen. It extends from a point just distal to the lesion at the base of the index finger obliquely downwards and outwards to terminate on the inner aspect of the ring finger.

them on the verandah of the patients' mess room, because he did not care much for the other attendants. The lepers often used to come near and talk to him while he had his meals. Food was cooked for the attendants and for the patients in the same kitchen. He used to wash his own utensils first, dry them on a special towel and then proceed to wash up for the patients. For about six months after he first went there a white leper used to kill and dress the sheep intended for and consumed by the staff and patients alike. There were four women patients. He had nothing to do with them. Of all the patients there during his term, that is from October, 1912, to May, 1917, he thinks only four men and three women died. They were buried in the cemetery in the lazaret. Only on one occasion did he burn one dead man's clothes and in one instance he helped to put the body in the coffin.

With two patients in particular he was very friendly. He even read their books and may have smoked their tobacco. On no occasion did he play card or other games with them. In May, 1917, he came to Sydney to a chemical job which six months later he lost for the usual reason. Since then he has been a wardsman in a hospital and a houseman in a private family.

When at Peel Island the patients often used to speak to him about their disease and one thing stuck in his mind because it was common to all of them. It was that before any signs of the disease appeared they all said they had had a period of feeling very lazy and run down, a sort of "don't care feeling"; they had to rouse themselves to do any work. He first noticed this feeling in himself in 1924 and there were no signs of leprosy on him then. If he had not heard the lepers speak of this lassitude *et cetera*, he would not have connected it with leprosy, but remembering this and the cut on his hand, he wondered and mentioned it to the doctor at the hospital where he was wardsman at the time. No notice was taken of it. This feeling lasted for some considerable time, over a year, and then in December, 1925, the first sign appeared on the back of the left hand, where he noticed a red-purple area about two and a half centimetres (one inch) in diameter. There was no irritation and he paid no attention to it until he thought of a leper he had seen with a similar spot and he tried to test it himself chiefly because it was near the cut on his hand. The hot bowl of his pipe was placed on the area and he did not feel it. He tried it again and even produced a blister and did not feel it, yet he felt it very much on the normal skin.

The red-purple area has gradually extended since then. Its present appearance is seen in Figure I. The next area noticed was over the styloid process of the ulna on the right wrist. This was three weeks later and the area was about the size of a one shilling piece. He then searched himself systematically all over and noticed a similar coloured patch on his left elbow. He found no other places, but for the last year he has noticed a lot of white spots, about the size of a pea, appearing on the back of his right hand (see Figure II.).

There is nothing of importance in the family history. His only other illness was pleurisy. He has not suffered from venereal disease. He came to my out-patient clinic at the Sydney Hospital on May 12, 1926, and his condition was diagnosed as leprosy. The areas on the left hand and right wrist were maculo-anæsthetic, while it was possible to feel the area on his left elbow, thus his infection seemed to be a mixed type. All the areas are anæsthetic and the anæsthesia in places goes a little beyond the coloured area. The ulnar nerve can be felt on each side, but it is doubtful whether it is thickened at all. I could find no other signs of leprosy. Slides were taken by pricking and *Bacillus lepræ* was not detected. A nasal swabbing also yielded no leprous bacilli. His serum failed to react to the Wassermann test on May 12, 1926. He was sent that day to the Coast Hospital. There, on account of the impossibility of demonstrating the *Bacillus lepræ* he has not been officially signed up as leprosy. Dr. Tebbutt has since demonstrated the causative organisms in a section taken from the left elbow.

The areas measure five by nine centimetres on the back of the left hand; two centimetres diameter on the left wrist; two and a half by two centimetres on the right wrist; four and a half by five and a half centimetres on the

FIGURE II.
Showing White Spots on Back of Right Hand and Macular Area over the Styloid Process.

left elbow. The centre of the lesions on the back of the left hand and on the left elbow are slightly whitish. The scars of the cut on the fingers can be seen distinctly, the red-purple colour is bordered by the cut on the middle finger and does not quite go up to the scar on the ring finger.

Conclusion.

I have shown that this case of leprosy is due to contact. The disease seems undoubtedly to have been contracted whilst the patient was an employee of the Queensland Government and thus the question of that State's *Workers' Compensation Act*, 1916, must be raised. In my opinion the patient is entitled to compensation and I will endeavour to obtain it for him in order that his future life in the lazaret may be made more comfortable.

Reviews.

GULLIBILITY.

A COLLECTION of essays reprinted from various periodicals on the most notorious examples of medical cults and charlatans in America and on strange health legislation and still stranger medical news in lay journals has recently been published under the attractive title of "The Medical Follies" and within four months has been reprinted four times.[1] Dr. Morris Fishbein, the renowned editor of *The Journal of the American Medical Association*, is the author of this instructive and enlightening volume. It is at once the most courageous, the most amusing, the most scathing and the most convincing exposure of cultism and of the gullibility of the public we have yet read. Direct attacks on charlatans and persons who profess and practise peculiar dogmas in medical treatment, are of little use to the community and are dangerous to the authors. Deliberate analyses of the origin and history of medical cults and quiet ridicule of the practitioners who tempt the public to believe in this form of pernicious nonsense, provide a surer means of arresting the trade. It is a sad commentary on human intelligence that the man who makes the most noise and who employs the most extravagant and impudent claims concerning his own superior attainments, is the one who can command the largest following. Orthodoxy rarely makes a strong appeal; heterodoxy seems to be irresistible to the majority. Dr. Fishbein has dug out the innermost secrets of the practice of Elisha Perkins, the man with the wonderful tractors, of Samuel Christian Friedrich Hahnemann, the founder of homœopathy, of Andrew Still, the originator of osteopathy, of D. D. Palmer, the inventor of chiropractic, of Albert Abrams, the man with the electronic apparatus, and of the many disciples of these successful charlatans. He is merciful in recognizing that some of them may have believed in the doctrines on which their practices were based, although he doubts whether the same honesty is manifested in the dealings of those who have attempted to keep the false doctrine alive after the downfall or death of the originator. He gives Hahnemann the credit of having stimulated the proper study of pharmacology. Notwithstanding the fact that his teaching and the whole doctrine of homœopathy are untenable and have been discredited, he certainly induced practitioners to deal individually with their patients and to refrain from prescribing excessive doses of powerful drugs.

Dr. Fishbein has a delightfully breezy way of telling his stories. Many of these stories are gems of wit and read like fairy tales. But they are true and in their context they provide the most damning evidence possible against the cults. It is with difficulty that we refrain from repeating some of them, but we do so because our object is to induce our readers to buy the book and read it from cover to cover. If ever any of these cults are introduced successfully into Australia, the wide distribution of Dr. Fishbein's book will suffice to enlighten the public and to render the experiment a failure. Perkins and his tractor are gone; we may have some similar form of quackery, such as a diagnostic planchette, a new magnet, but at present this kind of thing is not threatening. Osteopathy and chiropractic treatment have failed to hold their own in Australia. The fallacies have been exposed. Homœopathy is gradually dying; it has had its palmy days, but everyone knows that the successful homœopathic practitioner of today is one who does not confine his practice to homœopathy, save in imaginary illness and indisposition of a mild nature. The Abrams box is unlikely to survive, now that its founder has joined the majority. Dr. Fishbein completes the exposure of this remarkable hoax and concludes with the following statement: "Since time immemorial it has been known that a certain number of credulous persons will always be found who will believe anything that they cannot understand; this after all was the great discovery of Dr. Abrams. The complicated machinery that he devised for extracting the shekels of the unwary, was the *modus operandi* for putting his discovery to practical effect."

The author has some interesting facts to relate on the health legislation in certain American States. Some of our own legislation might also be held up to similar ridicule. He writes an admirable chapter on birth control and deals with this risky subject in an arresting fashion. The idiotic vapourings of the antivivisectionists receive the full force of his verbal artillery; not an argument is used that does not destroy the sophistry of the opponents of scientific experimentation. He has something worth while to say about the subject of rejuvenation. He adopts an eminently sensible attitude toward the doctrines of Voronoff and Steinach. There are several short chapters on the physical culture charlatans and so-called "big muscle boys." All these make good reading.

The penultimate chapter is entitled "Medicine and the Press." In this chapter he quotes some remarkable instances of errors in articles in lay newspapers dealing with medical matters. His pen is pitiless in exposing the ignorance of those responsible for the publication of this quasi-medical information. In Australia we suffer in the same manner. Many lay newspapers contain the most astounding statements concerning disease, research and the allied sciences. It would seem that the contents of cabled messages are reproduced as they are received without any intelligent criticism or control. Medical terms are usually spelt in an original manner and are often used in curious context. The person who obtains medical information from the Australian lay newspapers, is sadly misinformed.

"The Medical Follies" deserves a very wide circulation not only among medical practitioners, but also among the public. It has been written by an able American for Americans, but it will be found to be just as useful in any other country.

HERNIA.

FROM the title of Dr. L. F. Watson's book it will be seen that it contains a very full description of hernia.[1] It consists of six hundred and sixty pages and contains two hundred and thirty-two original illustrations which are excellently done. The book is well printed and easy to read and contains a full bibliography. Methods of anæsthesia are well described. All varieties of herniæ are described and their treatment in the case of the more usual forms fully discussed. Attention is drawn to "sliding herniæ" and also to the relationship of the bladder to both inguinal and femoral herniæ. The historical introduction is very interesting. To anyone needing a book for reference or a fuller description than that of the ordinary textbooks, this book can be recommended.

[1] "The Medical Follies," by Morris Fishbein, M.D.; 1925. New York: Boni and Liveright. Sydney: Angus and Robertson, Limited. Post 8vo., pp. 223. Price: 9s. net.

[1] "Hernia: Its Anatomy, Etiology, Symptoms, Diagnosis, Differential Diagnosis, Prognosis and Operative Treatment," by Leigh F. Watson, M.D.; 1924. St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 660, with two hundred and thirty-two original illustrations by W. C. Shepard. Price: $11.00.

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Medical Ethics.

The Federal Committee of the British Medical Association in Australia has just considered at the instance of the Queensland Branch a question connected with medical ethics of some importance. It appears that a medical practitioner C informed the Council of the Queensland Branch that a medical practitioner A advised his patient to enter a public hospital, presumably to have some treatment applied that A was not prepared to undertake in the patient's home. The patient did not follow the advice, but entered a private hospital where he was treated by another private medical practitioner B. C submitted his version of the happenings to the Branch Council in order that that body might determine whether or not B had acted unethically. The Council of the Queensland Branch dealt with this *ex parte* statement as if it were a hypothetical case on which a principle might be established, and expressed the opinion that when a medical practitioner refers his patient to a public hospital, he relinquishes his professional rights in respect of that patient. The complainant C apparently did not mean his charge to be regarded as the basis for the establishment of a general principle and therefore asked that the matter might be referred to some other body for adjudication. The Council of the Queensland Branch in addressing a specific question to the Federal Committee obviously adhered to its previous determination to deal with the matter as a hypothetical case.

The constitution of the British Medical Association provides for inquiries into charges of unethical conduct on the part of members. In Great Britain Divisions which have adopted rules of procedure in regard to charges of ethical misconduct, can undertake the preliminary inquiry and if it appears that the evidence is sufficient to substantiate the charge of conduct "detrimental to the honour and interest of the medical profession or calculated to bring the profession into disrepute," they may refer the matter to the Council for inquiry by the Ethical Committee. The overseas Branches have the powers of expulsion of members. Recently the South African Branches have requested the Association to alter its articles of association to invest federal councils with the same powers of expulsion as are exercisable by the Council of the Association and by the overseas Branches. The Federal Committee has not sought these powers and does not propose to seek them in the immediate future. Members should realize that when a charge of unethical conduct is laid against a member, the inquiry has to be conducted by the Council of the Branch or its ethical committee. If the member is found to be guilty of conduct deemed to be detrimental to the honour and interests of the medical profession or of the British Medical Association or calculated to bring the profession or Association into disrepute, the motion for his expulsion must be carried by a two-thirds majority at a general meeting of the Branch. There is no appeal within the British Medical Association against such a decision. The expelled member has his rights as a citizen and can seek his remedy in the ordinary courts of law. The judgement of the Privy Council in the case of G. S. Thompson *versus* the New South Wales Branch of the British Medical Association shows that if the procedure adopted is in accordance with the articles of association and the resolution for expulsion has not been influenced by malice, the courts of law will not interfere.

Any member of the Association may act as complainant in regard to an alleged act of unethical conduct on the part of another member. The Council of the Branch to whom the charge is submitted, must follow the proper procedure in its investigations of the charge. The charge must be communicated to the respondent and he must be given an opportunity to defend himself. The Council of the Queensland Branch would have acted improperly had it attempted to pass judgement without hearing the versions of B and of A. If it had been satisfied that the events as set out in the *ex parte* statement of C were indicative of a breach of ethical rules on the part of B, it could have called upon both A and B to consider the statement and to give

the Council an account of the incident each from his point of view. In the absence of such an investigation the Council could not deal with the statement as a charge.

Instead of following the course mentioned above, the Council of the Queensland Branch adopted the expedient of attempting to establish a general principle. It is usually a dangerous practice to establish a principle on an insecure basis. The Federal Committee recognized that under some circumstances a medical practitioner might deem it necessary to send his patient to a public hospital and yet be justified in expecting that the patient would return to him after his discharge from the hospital. On the other hand the patient might in other circumstances lose confidence in his doctor, if he failed to carry out the necessary treatment and advised that the treatment should be obtained in a hospital. A general principle could not be established in connexion with the specific question. Moreover, the statement of C could not be accepted as one of a hypothetical case. It contained a complaint of an alleged breach of ethical rules.

The lessons to be drawn from this incident are that the Federal Committee is not a court of ethical appeal, that a member charging another member with unethical conduct must be prepared to formulate his charges properly and to submit to the decisions of the Council of the Branch and lastly that it is eminently undesirable to introduce new general principles into the code of medical ethics.

Current Comment.

SELF-DIFFERENTIATION.

DIFFERENTIATION has been described as the fundamental phenomenon in development. The original germ cell must be regarded as essentially similar to any of the tissue cells of the body from which it has been derived, and division of the original cell takes place by mitosis in a manner similar to that occurring in the tissues of the adult body. When once segmentation has started, development proceeds in an orderly and regular fashion, the three primary layers are formed and from these the various systems take origin. It is not unnatural that hypotheses have been advanced to explain the extraordinary conformity to type which is observed. Why should a particular cell divide into other cells which grow in a particular direction? Why should one cell become more highly specialized than another? The two main views are those of the preformation theory and of the epigenetic theory. According to the preformation theory the embryo is predetermined by the predestination of certain areas or substances in the original cell for the formation of corresponding parts of the embryonic body. According to the epigenetic theory development is dependent on the occurrence of a regular sequence of stimuli.

The chick is the embryo best suited for embryological observations. Several observers have conducted investigations by means of grafts and by this means the process of differentiation has been studied. Among recent workers in this field is Dr. P. D. F. Murray. He has carried out a series of extensive observations on grafted limb buds.[1] Dr. Murray has previously worked on this subject in conjunction with Professor J. S. Huxley. Murray and Huxley concluded in 1925 that isolated fragments of the limb buds of the four day chick are able to undergo self-differentiation when living as grafts on the chorio-allantoic membrane of older chick embryos and that the bud of the four day chick is a "mosaic." They thought it likely that each of the morphological regions of the limb is represented by a mosaic and they expressed the opinion that no regeneration or regulation occurs in the fragments of the bud. At the same time they held that if a grafted fragment contains only part of a piece of the mosaic, that part can so regulate its future development as to form a complete morphological region corresponding to that of which it was originally a part. Dr. Murray claims that his present communication confirms most of the work of himself and Professor Huxley. As will be seen later he has modified the latter part of his earlier conclusions. It is unnecessary to give details of his technique or to attempt any description of the sixty-five grafting experiments which he carried out. It must suffice to state that when limb buds of the chick from three days of incubation onwards are grafted on to the chorio-allantoic membrane of older chick embryos, they are able to continue their growth and differentiation. The resemblance of the grafts depicted in the drawings to that seen in the normal chick embryo is striking. Dr. Murray sums the matter up by saying that the limb bud is a self-differentiating system. By self-differentiation he means a process wherein a part of an embryo developes from an earlier to a later stage without receiving from the remainder of the embryo any "formative stimuli" or any other assistance than a supply of food, oxygen, warmth, isotonic medium *et cetera*.

In discussing self-differentiation Dr. Murray raises questions of great interest to the morphologist. He points out that dependent differentiation is the opposite to self-differentiation. The example of dependent differentiation quoted by him is failure of the lens of the eye to develope in the absence of the optic cup. At the same time he points out that it is probable that all differentiation in the verte-

[1] *Proceedings of the Linnean Society of New South Wales*, Volume LI., Part 2, 1926.

brate embryo is dependent. He refers to the work of Spemann in regard to his "organizer of the first grade." The function of the organizer is determination and not differentiation. In other words the organizer is concerned with the decision into what parts of an adult particular parts of an embryo are to develope. Mangold found that if presumptive ectoderm cells of an embryo *Triton* which had attained a certain stage, were grafted among the mesodermal cells of another embryo, they would form mesodermal structures like somites. If, however, the presumptive ectodermal cells were taken at a later stage and treated similarly, they developed into ectodermal structures. In the latter instance the stage of determination has been passed. In other words the "determined" ectodermal cells have undergone self-differentiation. Even if it be the case that all development is ultimately dependent on Spemann's "organizer," it is not necessary to abandon belief in the self-differentiation of organs. Self-differentiation is merely the expression of the determination laid upon the cells at an earlier stage. When Dr. Murray states that all differentiation may in the last resort be dependent, he means that all "determination" is dependent. The location of the organizer is determined by the point of entrance of the spermatozoon and the latter is therefore regarded as fixing the position of the organizer as well as of the plane of symmetry.

Another of the many points of interest in Dr. Murray's investigations is his conclusion that limb buds at three, four and five days' incubation constitute a mosaic structure. He found that fragments developing as grafts would undergo self-differentiation to form those structures which they would form in normal development and no more. This conclusion differs from his previous findings with Professor Huxley and is subject to certain reservations. He discusses the question as to whether any two adjacent regions of the mosaic should be regarded as separated from another by a sharp dividing line or whether they should be regarded as shading off into one another so that between the two there would be an ill-defined "no man's land." The weight of evidence is in favour of the latter view. In one of the experiments the basal quarter of a bud gave rise to a complete femur and the next to apparently a complete tibia. Dr. Murray points out that the cells responsible for forming the ends should have been destroyed by the knife. Many of the difficulties disappear if the view is held that the boundaries between adjacent regions of the mosaic shade gradually into one another instead of being marked by hard and fast lines. It is not improbable that there exist between adjacent regions of the mosaic boundary zones of tissue capable of conversion into part of either of the two elements between which they lie, according to which of these regions gains control over them. It is well at this point to refer to the only instance of regulation found by Dr. Murray. Grafts were made from the longitudinal halves of a bud. Complete femora were formed, each of which had a rounded shaft. If regulation had not taken place each graft (grafts were examined nine days after implantation) would have contained the rudiments of a femur, resembling that bone split down the middle.

Dr. Murray's work is of interest when considered from the point of view of the preformation and epigenetic theories. If the limb buds consist of a series of mosaics, preformation in the limb bud, if not in the original germ cell, must be considered. If Spemann's "organizer of the first grade" be accepted, a complication in the way of holding a preformation view will be introduced. The organizer would probably be regarded as a "stimulus" to the original cell. In regard to the absence of "formative stimuli," postulated in Dr. Murray's definition of self-differentiation, it is probable that there is no influence of a nervous system. He points out that as far as is known there are no nerves in the chorio-allantoic membrane. He also refers to another point which is of more importance than the question of nervous influence. The graft is growing on a chorio-allantoic membrane eight days old: blood vessels are present in this membrane and it is supplied by hormones. If the graft were still attached to the parent stem its supply of hormones would probably be not so great or of so highly developed a quality.

THE SYDNEY CANCER RESEARCH FUND.

A FEW weeks ago we published an appeal to our readers for support in connexion with the endeavour of the University of Sydney Cancer Research Committee to collect the sum of £100,000. We asked the members of the medical profession to purchase one or more books of stamps at one pound for each book of twenty stamps. It is extremely disappointing to us that not one practitioner has responded to the appeal. There is much work to be done and there are many competent workers. The world is waiting for some genius to penetrate the shroud that conceals the cause of the diseases known as cancer. Some very valuable investigations in connexion with the problems of malignant disease have been carried out in Australia by Australian scientists. In every other country energy, ingenuity and money are being expended on a large scale for this purpose, but up to the present no one has succeeded in establishing the main facts of the ætiology and pathogenesis of carcinoma and sarcoma. The medical profession is known for its generosity. Need it be repeated that the general practitioner who comprises the major part of that profession, should evince an active interest in the progress of the work of his colleagues in the laboratory and should show that interest in a tangible manner. If the cause of cancer were discovered, the general practitioner of medicine would be a large gainer. He would be in a position to offer hope and help to his patients, while at the present time all that he can do is to submit them to a mutilating procedure which may result in the arrest of the disease, provided that the operation is undertaken before the process of metastasis has occurred. We ask our readers to send to the Editor of this journal one or more pounds to assist this important work.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Hydrogen Ion Concentration and Heart Volume.

H. GREMELS AND E. H. STARLING (*Journal of Physiology*, April, 1926) have studied the effect of changes of hydrogen ion concentration of the blood perfusing heart-lung preparations on the volume of the heart. They varied the pH of the blood by varying the carbon dioxide content of the inspired air. The lower the pH, the greater the volume of the heart. The dilatation affects both the systolic and the diastolic volumes. Addition of carbon dioxide to the air breathed caused dilatation of the coronary vessels and an increase in the coronary flow. The heart volume responds immediately to changes in pH of the blood. The behaviour of the heart volume during anoxæmia shows that up to a considerable degree of desaturation of the blood the heart still takes up sufficient oxygen for its requirements, partly by increasing the flow through the coronary vessels, partly by increasing the coefficient of utilization. A pronounced dilatation, often associated with heart block, sets in with oxygen saturations lower than 10%.

Pituitary Extract in Pregnancy.

H. H. KNAUS (*Journal of Physiology*, June, 1926) has examined the effect of pituitary extract on the pregnant uterus of the rabbit at different stages of pregnancy. During pregnancy the uterus reacts to pituitary extract in different ways at different stages and this might be due to a steady increase in the pituitrin secreted by the pituitary body or, even as Marshall and Dixon suggest, to a sudden increase of pituitary secretion at the end of pregnancy. Rabbits were kept under the influence of pituitary extract for periods of ten hours or so at different days of pregnancy. The duration of pregnancy in rabbits is fairly regular, thirty-one to thirty-two days. The minimum amount of pituitary needed to cause immediate parturition on the thirty-first or thirty-second day was 0·0075 milligramme of moist posterior lobe per kilogram of rabbit. This caused the immediate birth of one fœtus. On the thirtieth day not less than 0·3 milligramme of moist gland was needed. On the twenty-ninth day the minimum dose necessary was 0·6 milligramme. From the eighteenth to the twenty-eighth day no dose was found sufficient to cause abortion during the day on which it was administered, but the fœtuses were killed inside the uterus if the dose was sufficiently large and they were expelled some days later. From 1·5 to 2·0 milligrammes were necessary. From the first to the seventeenth day no dose was found which would disturb pregnancy. The interpretation of the experiments given is as follows. There is no increase of irritability or sensibility of the uterus during pregnancy, but, corresponding to the growth of the muscle cell, there is a regular rise of contractility of the muscle. The larger the muscle cell, the greater is its ability to shorten itself. During the first ten days of pregnancy the maximum contraction of each muscle fibre cannot produce a sufficiently great mechanical effect to influence the transport of ova or to disturb the connexion between the uterine wall and the placenta. The maximum shortening of the cells caused by the pituitary on the eighteenth day has just reached the threshold where the attachment of the placenta begins to suffer. The transition of the pituitary effect from the twenty-eighth to the twenty-ninth day of pregnancy is by no means as abrupt as may appear, for on the previous days there has been enormous devastation effected by the pituitary. On the twenty-ninth day the maximum contraction is just sufficient to empty the uterus, while in the last few days of pregnancy the uterus keeps on growing and increasing its contractility, so that a maximum dose of pituitary is no longer needed to produce the young. By the end of pregnancy the muscle fibres have become enlarged to such an extent and have acquired so great a power of contractility that their spontaneous contractions alone finally cause birth. There is no need to postulate any sudden influence or stimulus acting on the uterus at this time, as the labour pains are not essentially different in character from the contractions immediately preceding them, but only slightly more severe in degree.

Effect of Sleep on Respiration.

ALL investigators agree that there is a change in the rate and character of respiratory movements with the onset and during sleep. Some of these changes may be connected with the assumption of the horizontal position preparatory to going to sleep, but certain respiratory phenomena have been observed to be directly dependent upon passing from the waking to the sleeping state and a similar set of events, but in the reverse order, was observed upon waking. C. I. Reed and N. Kleitman (*American Journal of Physiology*, February, 1926) have made observations on respiratory periodicity during the onset of sleep and have reinvestigated the respiratory phenomena said to occur during sleep. Graphic records of the respiration of subjects were obtained before, during and after sleep. It was found that the respiratory rate was frequently increased when the subjects passed from wakefulness to sleep although in nearly as many cases there was no change in rate. Very slow normal rates were always increased. If the respiration was even and regular while the subject was awake, it usually became less regular during somnolence, but became regular again during sound sleep. The irregularity in respiration was commonly aperiodic, but sometimes resembled Cheyne-Stokes breathing. In the majority of experiments the amplitude of the abdominal excursions was absolutely decreased on the subject going to sleep and in a number of experiments the amplitude of the thoracic excursions was increased. The changes observed were not always the same in a given subject in successive experiments.

The Secretion of Pancreatic Juice.

SINCE 1902 the secretin hypothesis of Bayliss and Starling for the secretion of pancreatic juice has been generally accepted. This hypothesis states that secretin is derived from a precursor by the action of acid; prosecretin exists in that situation in which it is in a position to be acted upon by acid chyme and to discharge into the blood the substance which acts as a timely stimulus to the pancreatic cells. In a previous paper by Mellanby and Huggett it was shown that secretin exists in a preformed condition in the mucous membrane of the small intestine since active solutions of it may be obtained by solvents so diverse as water, 5% solution of sodium chloride, 0·85% solution of sodium chloride, 0·2% solution of hydrochloric acid, 0·1% solution of sodium hydroxide, 75% solution of alcohol and 75% solution of acetone. It is evident, therefore, that there exists no immediate causal relation between gastric acidity and pancreatic secretion. J. Mellanby (*Journal of Physiology*, June, 1926) has demonstrated another mechanism for the passage of secretin into the blood. He finds that the introduction of bile of an adequate reaction into the duodenum of a cat causes a copious secretion of pancreatic juice. The active substance present in bile is cholic acid, but the activity of cholic acid is profoundly modified by (i.) its association with taurine and glycine and (ii.) the presence of mucin in the bile. The optimum reaction of bile as a pancreatic stimulant varies with the state of digestion of the animal. In a fasting cat the optimum reaction is pH 7·8; in a cat in which gastric digestion is actively proceeding, the optimum reaction is pH 6·5. Paralysis of the vagus by atropine or of the motor side of the sympathetic by ergotamine does not diminish the capacity of bile to act as an alimentary stimulus for the secretion of pancreatic juice. The immediate stimulus for the secretion of pancreatic juice is secretin. Secretin contained in the cells of the intestinal mucosa is carried into the portal blood associated with the bile salts contained in the fluid absorbed from the intestine. The facts offer an adequate basis for the appreciation of the severe digestive disturbances which occur in catarrhal jaundice.

Blood Coagulation with Tissue Extracts.

IT has been known for many years that bruised or broken tissue introduced into the blood stream causes intravascular clotting. Wooldridge

found that extracts of tissues had the same effect and gave the name of "tissue-fibrinogen" to the active material of the extract. H. E. Burke and J. Tait (*Quarterly Journal of Experimental Physiology*, April, 1926) have studied the effects of such tissue extracts on blood platelets and the relation of these to clotting. The extracts used were saline extracts of the ground tissues previously freed from blood. The active component is not in true solution, but is bound up with material in suspension. Any injection of active tissue extract causes a prompt fall in the number of circulating platelets. The number soon rises again, to fall once more on repetition of the injection. Negative phase blood produced by repeated slow injection of the tissue extract may behave anomalously when one attempts to defibrinate it by whipping it with a stick. If the platelets are considerably reduced in number, no fibrin collects, but the blood eventually clots in lumps after a long delay. When a starved animal is killed by an injection of a minimmum lethal dose of active tissue extract, death is due to formation of a platelet thrombus in the right ventricle and pulmonary artery. This thrombus is similar to that described by Welsh as occurring sometimes in pneumonia. Blood from which platelets have been virtually removed by tissue extract injections still contains fibrinogen and will clot firmly on addition of tissue extract. This blood does not spontaneously clot.

BIOLOGICAL CHEMISTRY.

Action of Irradiated Sawdust.

N. S. Lucas (*Biochemical Journal*, November, 1925) has made a study of the action on a photographic plate of sawdust and cholesterol irradiated by a mercury vapour quartz lamp. It had already been shown in the Lister Institute that pine sawdust, exposed to the radiations of a mercury vapour quartz lamp, developed antirhachitic qualities. For some time it had been thought that this observation was due to secondary radiations or some emanations given out by the particles of wood exposed to the ultraviolet rays. This view was countenanced by the statement that cod liver oil, egg yolk and other antirhachitic substances had an action upon a photographic plate, even when the substances were covered by a quartz plate sealed so as to preclude the action of a volatile substance. It has long been known that similar effects on photographic plates may be due to the evolution of hydrogen peroxide. The author's investigations have led him to conclude that the photographic action of pine sawdust is due to its slow generation of hydrogen peroxide. The author records the result of an experiment in which the emanation from sawdust was drawn along a blackened tube and allowed to fall on a photographic plate. A shadow was observed on the plate extending as a fan in the direction of the moving current of air. A series of studies have been made upon the photographic action of irradiated cholesterol. The author concludes that all photographic action from irradiated cholesterol can be prevented if a plate of quartz be arranged to exclude effectually all vapour from the photographic plate.

Vitamins in Preserved Eggs.

E. Tso (*Biochemical Journal*, November, 1925) has made a study of the vitamins in eggs which have been chemically preserved. The Chinese eggs known as "pidan" are fresh ducks' eggs which are coated with a paste made from soda, straw ash, table salt and lime; they are then stored in sealed earthenware jars. Preservation is effected and the eggs are ready for use as "pidan" in about one month. Such eggs have a fresh piquant lime taste and an ammoniacal odour when freshly opened. Analyses of such eggs have shown a distinct increase in ash and in alkalinity, together with partial decomposition of proteins and phospholipins. Four sets of experiments upon rats have shown that fat soluble Vitamin *A* is abundantly present. Small quantities of the yolks of treated eggs have readily restored health to rats suffering from a deficiency of Vitamin *A*. Again, rats receiving an oxidized ethereal extract of the yolks of these eggs as the source of Vitamin *A* have soon developed xerophthalmia and lost weight, but have been restored to health by the use of non-oxidized ethereal extract of "pidan" yolks. Six rats which had grown vigorously on a normal diet, have been given a basal ration deficient in water-soluble Vitamin *B*. As soon as they commenced to lose weight they have been given preserved egg. They have shown no improvement and in time some have died. One rat made a rapid recovery when given the yolk of fresh ducks' eggs. It would appear that the rich store of Vitamin *B* in ducks' eggs is destroyed during the process of preservation. Two sets of experiments have been used to investigate the antirhachitic value of "pidan." These experiments show that the antirhachitic food factor like the growth promoting and antixerophthalmic vitamin or Vitamin *A* is not affected to any appreciable extent by the chemical changes in the preserved eggs.

Cyanates in Blood.

E. Gottlieb (*Biochemical Journal*, November, 1925) has made an investigation concerning the presence of cyanates in blood. Since cyanates and cyanic acid might be precursors of urea in the blood or of ammonia in the kidneys in accordance with the observations of Werner that two molecules of cyanic acid and one molecule of ammonium cyanate in neutral or alkaline reaction are condensed to urea and that cyanic acid in acid solution is hydrolysed to ammonia and carbon dioxide, a number of observers have stated that blood contains appreciable amounts of cyanates. Thus Montgomery has asserted that the blood of rabbits and of cats contains 0·8 to 1·0 milligramme cyanic acid per hundred cubic centimetres of blood. The author has determined the production of ammonia in samples of blood divided into two portions. One part is kept distinctly acid and the other part faintly alkaline. When small amounts of cyanate are added to such samples of blood, there is distinctly more ammonia produced in the acid samples than in the alkaline portions. Samples of blood and of plasma from normal animals show no variation in the production of ammonia, whether the samples be kept acid or alkaline. From these and analogous experiments the author concludes that blood cannot contain cyanates in greater concentration than 0·1 milligramme per hundred cubic centimetres of blood. Some further observations have been made upon the effects of running solutions of cyanates slowly but continuously into the veins of rabbits and dogs. The author finds that solutions are readily toxic even when such small amounts as 0·1 milligramme to 0·5 milligramme of cyanate per kilogram per hours are administered. Estimations of the amount of cyanate introduced and of the amount of cyanate in the various parts of the body show no sign of any destruction of cyanates in the course of one hour, the usual duration of the experiment.

Uric Acid Retention.

W. G. Lennox (*Journal of Biological Chemistry*, December, 1925) has made a study of the retention of uric acid during fasting. He has measured the amounts of uric acid in the blood and in the urine during twenty-one fasting periods of healthy male persons of ages from fifteen to thirty-eight years, the fasts being of different durations. The estimations have been made daily. In different individuals estimations have been also made of the non-protein nitrogen in the blood, amino acid nitrogen in blood, plasma bicarbonate, urinary hydrogen ion concentrations and urinary total nitrogen. The author shows that the high concentrations in the blood have been due to retention of uric acid. The usual diurnal variations in excretion disappear. It would appear that retention can first be observed in the plasma. Feeding the subject with fats has not lessened the retention, whereas feeding with carbohydrate, thyreoid extract, amino acids and protein has increased elimination. It seems evident that variations in the excretion of uric acid following the use of various purin-free diets do not mean variations in the production of uric acid, but variations in the amounts contained in the blood and tissues. The author suggests the use of diets with a low content of fats in the treatment of gout.

British Medical Association News.

MEDICO-POLITICAL.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on August 13, 1926, DR. R. J. MILLARD, C.M.G., C.B.E., the President, in the chair.

Treatment of Insured Injured Workers.

DR. R. H. TODD moved on behalf of the Council that no member of the Branch should accept a salary from any insurer or employer for the treatment of injured workers insured under the *Workers' Compensation Act*, 1926, but that the remuneration for the treatment should be by fee. He stated that it was important to take steps to insure that the interests of medical practitioners in connexion with the new Act were not overlooked. The term injury as used in the Act covered diseases other than venereal diseases, provided that the employment was a contributory factor. It was obvious that a great deal of treatment would be required, since the employer was required to provide it. The Act introduced a novel principle. The employer was entitled to supply the treatment, even if the injured worker had his own medical attendant. It was evident that advantage would be taken of this clause if it saved the employer or the insurer money. Dr. Todd proceeded to explain the clauses of the Act in some detail. He regarded the Act as a bad one, but he anticipated that after a time of trial, it would need amendment. He referred to the meetings that had been held by the affiliated local associations throughout the State to discuss the Act and its significance to the medical profession. The Branch had always opposed payment by salary for treatment of any kind. In the instance before them it was particularly necessary for the medical profession to protect itself against exploitation. It had come to his knowledge that medical officers engaged by insurance companies were approaching other practitioners to work under them in a salaried position. He thought that it would be advisable to delete the words "from any insurer or employer" from the motion, so that it would read:

> That no member of the Association shall accept a salary for the treatment of injured workers (*Workers' Compensation Act*, 1926) and that in respect of all treatment of injured workers remuneration for services shall be by fees as is customary in private practice and not otherwise.

DR. L. L. HOLLAND stated that he had been working with Dr. D. H. Graham who held a position in connexion with an insurance company. There was no contract for treatment. The treatment was usually carried out by the patient's own local practitioner.

DR. H. E. LEE also spoke on this aspect of the subject. Dr. A. Leiper had undertaken not to carry out treatment and not to engage other medical practitioners to do this work.

DR. R. P. WAUGH said that it resolved itself into the question of the establishment of precedent. In principle all would agree that salaries should not be accepted. He noted that Dr. Leiper had undertaken not to treat injured workers in his official capacity. But there was nothing to prevent him from engaging in general practice and in his private capacity he would have an advantage over other medical practitioners in regard to this work.

DR. A. M. STANTON moved that the words "as is customary" be amended to read "on the same scale as is customary." He wished to prevent a lower scale of fees than those obtaining in private practice in the district from being adopted for work under the Act.

The amendment was seconded by DR. J. KERR.

DR. R. H. TODD stated that the matter was determined by the provisions of the Act. He did not think that the amendment was necessary.

DR. R. J. MILLARD referred to the steps that had been taken to prevent practitioners from charging excessive fees. The amendment safeguarded them from being paid at too low a rate.

The amendment was carried.

DR. J. I. PARER spoke of his employment at a large industrial works. He was paid at a fee per visit. If the injured worker had his own attendant and cared to go to him, he was encouraged to do so. He spoke of the difficulty in determining what was a fair rate of remuneration for this class of work.

DR. S. SHELDON thought that the Council of the Branch should have considered the bill while it was before Parliament. They seemed to forget that the employer was the man who had to pay. The actuaries wanted to know what the medical rates would be. He could not understand the difference between accepting a salary for lodge work and accepting a salary for the treatment of injured workers.

It was pointed out to Dr. Sheldon that there was a great difference between a capitation fee and a salary.

Continuing Dr. Sheldon stated that Government medical officers received fees for treatment. In these circumstances he recommended caution and the avoidance of any hurried conclusion. The control of the whole matter might pass into the hands of the Government Insurance Office. But even if other companies were acting on behalf of the employers, the insurer would always have the actual decision.

DR. J. KEARNEY did not agree with Dr. Sheldon's attitude. He congratulated the Council for taking action. The insurance companies wanted too much; this meant that the medical practitioner got too little.

DR. C. H. E. LAWES held that Dr. Sheldon was under a misconception. The Council had a duty to the members and dared not adopt a waiting attitude. There was extreme urgency. The Branch objected to salaries, especially for an undefined amount of work. He trusted that the profession would show that it was united and determined to adhere to its principles.

PROFESSOR F. P. SANDES said that if the proposition were adopted, they would want to know when it would become operative, how the Council proposed to enforce it and what they were prepared to do with the medical officers of the insurance companies. The patient had the right to choose his medical attendant. He, the speaker, was satisfied that the Government Insurance Office was prepared to take every advantage that presented itself. The Branch should do all that lay in its power to compel injured workers to obtain treatment by their own medical attendants.

DR. J. C. STOREY, O.B.E., dealt with the question from three points of view. In the first place there was the point of view of the injured worker; in the second place there was the point of view of the employer and in the third there was the point of view of the medical profession. The injured worker had the right to choose his own doctor to attend him. Since the employer had to pay, he certainly had a moral right to influence the choice. The Act gave him the legal right to provide a medical attendant. In practice the matter would probably be one of mutual arrangement. To be logical the insurance companies should have the privilege of selection of doctor. This might reasonably be extended to the choice of a consultant. Dr. Storey maintained that ordinary fees should be charged by practitioners attending injured workers.

Dr. T. W. Lipscomb insisted that there was no time to lose. He disagreed with Dr. Sheldon who advocated a policy of caution and procrastination.

DR. C. BADHAM did not think that there was any new principle involved in the *Workers' Compensation Act*. The industrial medical service was gradually being extended. It was essential that industrial work should be increased and that the health of the workers should be safeguarded as carefully as possible. The fees for this kind of work must be adequate. On the other hand, high rates might

have the effect of limiting the amount of work done and this would be bad for the community and bad for the profession.

In his reply Dr. Todd informed the members that the Council had appointed a subcommittee to prevent medical practitioners from charging excessive fees for attendance on injured insured workers. He was satisfied that the motion, if carried, should become operative without loss of time. If the patient received treatment in hospital, the doctor would not benefit unless some special arrangement were made. In reply to Dr. Badham he pointed out that the industrial medical officer undertook only first aid treatment. The medical practitioner would have to give continuous treatment to injured workers under the Act.

The motion as amended was put to the meeting and was carried.

DR. RICHARD ARTHUR, M.L.A., was granted leave to introduce another matter in connexion with the *Workers Compensation Act* and to give notice of motion. After sketching the history of compensation for workmen injured in the course of their occupation in New South Wales up to the passing of the new Act, he pointed out that injured workers were entitled to treatment in hospitals. The Government was endeavouring to compel all injured workers to enter hospitals. He regarded this as a scandalous thing. The hospitals had allowed themselves to be stampeded into a temporary arrangement whereby 600,000 out of a total of 900,000 breadwinners were asked to use the public hospitals. The injured worker should be treated by the private practitioner. The medical profession would be insane if it did not insist on its rights. He therefore asked that the whole question of treatment of injured insured workers in public hospitals be discussed at a special meeting.

The members assented to this proposal without further discussion.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Balzer, John Camfield, M.B., Ch.M., 1926 (Univ. Sydney), Mater Misericordiæ Hospital, North Sydney.

Rorke, Frederick Charles, M.B., Ch.M., 1926 (Univ. Sydney), Port Kembla.

Wheelihan, John Maurice, M.B., Ch.M., 1926 (Univ. Sydney), Clyro, Victoria Road, Bellevue Hill.

Davis, Keith Joseph Brandon, M.B., Ch.M., 1926 (Univ. Sydney), 34, Gowrie Avenue, Waverley.

Dickson, Ian Thomas, M.B., Ch.M., 1926 (Univ. Sydney), Rabaul, New Guinea.

Oag, Hugh Stewart, M.B., Ch.M., 1925 (Univ. Sydney), Campbell Street, Hunter's Hill.

Peck, Grace Jean, M.B., Ch.M., 1926 (Univ. Sydney), Henry Street, Gordon.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

O'Collins, Joseph Bernard, M.B., B.S., 1926 (Univ. Melbourne), Albert Park, East Melbourne.

Hamilton, James Joseph, M.B., B.S., 1926 (Univ. Melbourne), Homœopathic Hospital, Melbourne.

Counsell, Walter Duff, M.B., B.S., 1926 (Univ. Melbourne), 3, Foster Street, St. Kilda.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the Adelaide University on July 2, 1926.

Influenzal Pneumonia.

DR. F. BEARE said that patients suffering from epidemic influenza with pneumonia who did not exhibit an increase in rate of respiration in the first fourty-eight hours, although temperature was raised, had other distinctive features, namely that they coughed up pure blood and the onset was accompanied by stabbing pain in the thorax. Examination then showed no fever or increase in pulse rate or in respiration, but the next day both temperature and pulse rate increased, but not respiration. He was of opinion that the condition in these cases commenced as a pleurisy and spread inwards. He asked: (i.) Why was there an increased respiratory rate? The pain was not the cause. (ii.) Was there any theory about the pathology of the onset? (iii.) Did pneumonia start from within or *vice versa*?

DR. A. J. LEWIS asked might not these cases be due to infarcts? DR. BEARE inquired as to the possible origin of the infarcts.

PROFESSOR J. BURTON CLELAND pointed out that the influenzal pneumonia of the last epidemic was characterized by many infarcts and that the patients had very little respiratory distress.

Herpes and Varicella.

Professor Cleland reported another instance of one member of a family having herpes, followed by another having chicken-pox. He stated that Dr. F. Cox, of Helensburgh, New South Wales, had recently seen a man with a typical chicken-pox rash. On being asked if he had been away or had any visitors with a similar rash, his wife said that the two children had had the same thing and she herself had had a rash on the lower right ribs. Her herpes had nearly dried up and probably had developed about five weeks previously.

DR. A. A. LENDON reported a case of a father with herpes and a child with chicken-pox.

Corpus Luteum and Pregnancy.

Professor Cleland exhibited a specimen from a woman, aged twenty-eight years, admitted to the Adelaide Hospital with a diagnosis of meningitis. On examination she had been found to have clenching of the jaws with retraction of the head and stiffness of the neck. The condition clinically was not like tetanus. At the *post mortem* examination she had been found to have a small fœtus in the uterus and a cystic *corpus luteum* in one ovary. He pointed out that in cows, he was informed, a cystic *corpus luteum* rendered the animal sterile. It was a question in this case whether this *corpus luteum* was old or whether it was associated with the pregnancy. From its structure it was apparently an old one. But this did not explain the tetanic symptoms the patient had manifested with the pregnancy. He was of opinion that the explanation might be the detachment of fœtal cells with their carriage by the blood stream to the brain and lodgement therein, these producing the symptoms. He quoted a patient of Dr. Ray who was supposed to be suffering from tetanus, but whose condition was found to be toxæmia of pregnancy.

DR. A. A. LENDON stated that experiments on animals indicated that excision of the *corpus luteum* caused abortion, whereas Dr. W. A. Verco had told him that he had excised the *corpus luteum* in a pregnant woman and abortion had not occurred.

Cystic Disease of the Kidneys.

Professor Cleland also exhibited specimens of kidneys with innumerable small cysts. Apparently they were con-

genital polycystic kidneys in which the cysts had not reached a large scale. He stated that these cysts were often associated with cystic disease of the liver or pancreas. The small mass of cysts the size of a walnut had been attached to the ileum. On section they had lost all their epithelial lining. The patient had also suffered from tuberculous ulcers in the bowel.

Correspondence.

THE WORKERS' COMPENSATION ACT.

SIR: At a meeting of the Central Western Medical Association held last night the *Compensation Act* was discussed at length as far as the general practitioner is affected, but the matter of hospital practice was deferred, as being a different matter.

Now it appears to me that the whole question of hospital practice is of vital interest not only to the members of the honorary staffs, but to the general practitioner. What is to present the insurers paying a certain subsidy, *per capita* if you like, and sending all their cases to the hospitals for treatment.

Dr. Todd in the course of his remarks said that the old idea that hospitals were only for the indigent poor had gone by the board, but we in Newcastle have been fighting strenuously against that impression. We do not consider that, acting in an honorary capacity, we should be called on to give our services to people who can well afford to pay for those services. Now that the new *Act* is in force are we to continue to give our services free to a group that is required by law to pay up to fifty pounds per man for treatment and in doing so deprive the practitioner who is not on the staff of his means of livelihood?

Dr. Todd said that this *Act* was largely modelled on the American system. In that case why not adopt their hospital system as well, charges and all?

I must admit that the man not on the staff would still suffer, but the feelings of the fortunate member of the staff would no longer be hurt by the sight of an injured man in receipt of a salary of £749 being treated by him free.

Yours, etc.,

W. R. BEESTON.

20, Bolton Street, Newcastle
New South Wales,
August 7, 1926.

SIR: *Re* "Compensation and Injury to the Back" Dr. E. C. Chisholm's letter (THE MEDICAL JOURNAL OF AUSTRALIA, September 4, 1926) prompts a comment. All will agree with the difficulty in deciding whether or no malingering or the more common exaggeration of disability, is present or not. Nevertheless if one tries to get a clear mental picture of the possible pathological condition it is a great help in coming to a decision. In this respect I would criticize the statements contained in the letter in question. Dr. Chisholm considers the painful back as "a fibrositis involving the nerve sheaths and often associated with true rheumatism of the lumbar muscles." As a definition this does not help much. What is true rheumatism of a muscle? Again "the characteristic attitude of the back bent at the lumbo-sacral joint" is a faulty description. Acute lumbar fibrositis of whatever origin is a very definite pathological state with very definite pain and very definite signs. Of these the most obvious is the characteristic attitude. On account of the exquisite pain produced by any movement involving the inflamed fibrous tissue, reflex protective muscular spasm fixes the lumbar spine in the position of greatest ease, that is one which varies slightly with the site and magnitude of the involvement, but usually one in which the normal lumbar curve is somewhat undone, that is a flattening of the lumbar spine. This throws the body weight forward a little and hence flexion—not at the lumbo-sacral joint, but at the hips and knees—in order to preserve balance.

He states: ". . . in the majority of genuine cases, there being no objective signs whatever . . ." Again one must disagree. The genuine case offers very definite signs. The malingerer tries to imitate the rigidity *et cetera* and on his skill in so doing and on the patience and the guile of the doctor in attempting to get him to perform some act impossible in a genuine "lumbago" depends to a large extent his detection.

In a railway construction camp Dr. Chisholm was working under difficulties and one must appreciate the probable absence of the valuable aid to diagnosis given by X rays. A doubtful case of malingering may be cleared up by raying in two positions: (i.) with hips as fully flexed as possible and (ii.) with hips extended. The malingerer in the strange surroundings of the ray room may allow himself to be trapped into undoing his assumed lumbar rigidity and the two rays are evidence of this.

In diagnosis of back injuries the use of X ray is indispensable, but we are dealing now only with the question of whether the claimant with the strained back is genuine or not.

Yours, etc.,

J. HOETS.

143, Macquarie Street, Sydney,
September 6, 1926.

ENUCLEATION OF TONSILS.

SIR: I am in accord generally with Mr. Watkins's letter rearding tonsils and adenoids and I cannot believe that Mr. Murphy intends us to take him seriously.

In the present state of our knowledge there appears only one method of treatment of tonsils and adenoids when they are causing trouble and that is by surgical removal. Whether the removal of adenoids in the early stages would make the plain women of whom Mr. Watkins speaks, beautiful, I cannot say. But such cases cannot be many. I have been under the impression that Australia possessed few such women.

Now regarding his wholesale condemnation of the "prehistoric instrument," the guillotine, no matter what method is employed or what instruments are used, it is of no consequence provided that the objectives are obtained without any damage to the neighbouring parts and that there are no after complications that can be reasonably avoided. Some men get better results by one method and some by another, but to condemn either is entirely wrong. Let each follow the method that suits him best. All cases are not suitable for the use of the guillotine, but probably 90% are, and in competent hands a perfect enucleation can be attained. It is essential for good work that one uses intratracheal anæsthesia and a blunt guillotine. Any bleeding points are secured just as in a dissection operation.

Some of the advantages of a guillotine operation are: It is an easy operation after a little practice; it is a more rapid operation than by dissection; there are no greater risks or complications by this method compared with dissection. And lastly in properly selected cases the results are perfect.

I believe that a badly executed tonsillectomy by dissection does greater damage to the faucial pillars than does a badly executed operation by the guillotine.

Yours, etc.,

JAMES M. BAXTER, M.D. (Melb.),
Surgeon to Ear, Nose and Throat Department,
Saint Vincent's Hospital, Melbourne.

Lister House, Collins Street, Melbourne,
August 10, 1926.

SIR: Dr. Hennessy at the August meeting of the Victorian Branch described my tonsil surgery as bad and my mortality rate as high. In your issue of September 4 he pays me a great compliment by describing a new method of tonsillectomy which I have performed for many years, with some new addition of which I strongly disapprove and with retention of other details which I have discarded.

(i.) Position of patient. Head dorsiflexed and lowered is the best for the formation of a large blood clot and was responsible for the only death I have had about thirty-three years ago.

(ii.) Lifting up of the anterior pillar of the tonsil should proceed from below where it is seldom adherent to the tonsil.

(iii.) The best grasping instrument to hold the tonsil is a blunt guillotine. A volsellum is useless and should only be used in those rare cases where one fails to grip the tonsil with a guillotine. As soon as the anterior pillar is released I apply a guillotine.

(iv.) Special instruments are not required. If the anterior pillar is strongly adherent, I snip through the adhesions with a long pair of scissors curved on the flat. For lifting off the tonsil I use a sharp elevator like the one figured in Dan McKenzie's book. It should be fairly sharp. After freeing the body of the tonsil, clip the upper group of vessels with artery forceps and then dislodge the upper pole with the curved end of McKenzie's elevator.

(v.) Hæmorrhage is most profuse in uncomplicated cases from the upper set of vessels and most persistent from the raw surface after removal of lower lobe. If the posterior pillar be torn or cut through very alarming hæmorrhage may arise from the damage to the large veins running parallel to it. These are the only vessels I have ever ligatured.

Now as regards my mortality rate referred to by Dr. Hennessy, I don't know how many cases I have done. I should think well over thirty thousand. For thirty years, I was constantly operating at two hospitals and for many years at three hospitals and when I had a large lodge practice I often did ten cases a week in private.

Case Number 1, 1899, stout female, *ætatis* forty-two, head dorsiflexed and chloroform anæsthesia. Inhaled a large blood clot and died instantly.

Case Number 2, 1914, boy, *ætatis* fifteen, gross case of congenital syphilis. Died under ethyl chloride anæsthesia; Melbourne Hospital.

Case Number 3, medical practitioner. Died two hours after attempted removal of stump of tonsil with peritonsillitis. Patient collapsed five days previously and had had many attacks of peritonsillitis. He stopped breathing after a few whiffs of anæsthetic. Tracheotomy performed and revived patient. His epiglottis and laryngeal mucosa were enormously thickened. We massaged his heart and tried every other means to resuscitate him but failed. He certainly had septic laryngitis and probably pericarditis as well.

In thirty-six years of practice I have only had one death directly attributable to this operation and that was because I used the dorsiflexed head position then in vogue. I am glad I can agree with Dr. Hennessy on two points: (i.) The wickedness of the crushing cutting guillotine and (ii.) the unnecessary use of intratracheal anæsthesia; I understand several deaths have occurred in Melbourne through its use.

Every surgeon likes his own method best and the operation he should perform is that which appeals to him most. For over ten years I have stuck to this method I have described above to the exclusion of all others. The difficult question with regard to tonsils is when to operate; it is easy enough to advise every case that comes along to have a tonsillectomy.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street, Melbourne.
Undated.

"SEPTIC" TONSILS.

SIR: I did not intend to be drawn into the argument as to what constituted "septic" tonsils, but Dr. Stewart's direct request with thanks in advance forces my hand.

The word septic is derived from a Greek verb meaning to make putrid and if used at all in medicine, apart from public health, should be reserved for pyogenic organisms and not applied to organs or tissues. Even in connexion with organisms I refrain from using the word, for it is used too loosely and if used at all should be allowed to apply to the organisms of tuberculosis, glanders, actinomycosis *et cetera* as well as to the commoner pyogenic organisms.

The diagnosis of chronic tonsillitis is sometimes very easy and at other times impossible. Patients with recurrent tonsillitis may in the intervals between attacks have small mobile tonsils, without surrounding congestion and without *débris*, which can be massaged from the crypts, and yet the patient's general health is so much improved by removal that there can be no doubt that there was a chronic infection causing general symptoms in the intervals. One must not go too much on the appearance of the tonsils, for those that appear unhealthy, may be found on microscopic examination to be healthy, whilst latent tubercle or abscesses may be found in tonsils which appear healthy. However, changes in the appearance of the tonsils are of great import and I cannot lay down a better rule than: When in doubt, and there is time for delay, watch the tonsils. In no branch of medicine is more experience and intuition required than in condemning tonsils. The personal element enters into it very much and it is difficult to put into words the little points which bias one one way or the other.

I can only condemn the practice of advising tonsillectomy whenever streptococci can be cultured from a tonsil swabbing regardless of history or other signs. Short chain streptococci, if found, may be normal inhabitants of the saliva. Long chain streptococci may not have come from the tonsil, but have got washed there from any focus of infection in the mouth, nose or pharynx. Also negative findings for streptococci do not exclude their being present deep in the crypts or even in the tonsillar tissue. Again other organisms besides streptococci cause chronic tonsillitis. However, I feel that when advance is made, it will be along bacteriological lines.

I append the indications I use for tonsillectomy for conditions other than neoplasm:

(*a*) Cases causing respiratory obstruction. Usually nasal obstruction with enlarged tonsils is due to coexistent adenoids. But I have met with all degrees of obstruction due to enlarged tonsils and in the March 6 issue of this journal this year published a case where emergency tracheotomy was required.

(*b*) Cases where the tonsils are more than very slightly enlarged where there are indications for removal of adenoids. The frequency with which tonsillectomy is necessary after adenectomy makes this rule advisable.

(*c*) Cases where there has been more than one peritonsillar abscess, for nearly all these cases have more tonsillar trouble later, unless radically treated.

(*d*) Cases where the tonsils are under suspicion and there is a chronic lymphadenitis of the tonsillar group of cervical glands (whether due to tubercle, commoner pyogenic organisms or a mixed infection) and where dental and accessory air sinus infections have been excluded.

(*e*) Cases of chronic catarrhal deafness with a useful degree of hearing remaining, where the tonsils are the least under suspicion, except in the aged or very debilitated. Nasal obstructions and nasal and accessory sinus infections are dealt with also. This rule sacrifices some tonsils unnecessarily, but this is justifiable, for by such treatment the progress of an otherwise incurable and progressive disease is almost always apparently slowed, is usually stopped, and occasionally actual improvement in hearing results.

(*f*) Cases of continued recurrent sore throats with definite changes in the tonsils during attacks. If the attacks are almost continuous and are also very subacute, any dental or gross sinus infection should receive attention first, as this may suffice.

(*g*) Cases where the tonsil is under the slightest suspicion and there is known to be an area of focal infection in the body and the common foci have been excluded. Such an examination should include a urine examination,

X ray of the teeth and a prostatic smear. The presence of a focus of infection may be assumed in cases of fibrositis and osteoarthritis (especially when of the rheumatoid type) and must be suspected in such conditions as endocarditis of doubtful cause, secondary anæmias, certain skin diseases and cases of exophthalmic goitre. In the latter disease remarkable cures have followed tonsillectomy. In one case a patient nearly lost her life with acute hypothyreoidism after the stimulating toxin was removed and was apparently saved only by the administration of thyreoid extract for a few weeks.

(*h*) Similar cases to (*g*) where the tonsils are, as far as I can tell from the history and physical signs, healthy, but where the physician in charge asks me to remove the tonsils. If a very thorough general examination has been carried out first and no other focus found, I am willing to perform tonsillectomy, provided the patient knows the treatment is in the nature of an experiment. If, however, the case permits I prefer to watch the tonsils for a period first.

(*i*) Cases of chronic lacunar tonsillitis, but I do not remove tonsils with *débris* in only a few crypts if there are no other symptoms.

(*j*) Cases of chronic otorrhœa where there is the least suspicion of tonsillar trouble. Such treatment has saved a number of major operations.

(*k*) Cases complaining of of cachosmia where there are no dental cavities (interstitial ones are the most important here), no chronic discharging apical sinuses and there is no atrophic rhinitis and no infection of the antra or ethmoidal cells, but where *débris* can be expressed from tonsillar crypts and the patient recognizes the odour of the *débris* as that of which he is complaining.

There may be other indications which I cannot call to mind unless I look back through a mass of case cards, but I think the above is fairly complete.

It will be seen that the indications as given above are empirical, unscientific and compared with many branches of modern medicine deplorably unsatisfactory. However, they are the best I can give and if anyone can put the whole matter on a scientific basis, he will have my heartiest thanks and deserve that of a large proportion of the inhabitants of the globe.

Yours, etc.,

A. B. K. WATKINS,

Honorary Ear, Nose and Throat Surgeon to the Newcastle General Hospital.

September 4, 1926.

HIGH TENSION IN IRITIS.

SIR: In your issue of August 7 Dr. Temple Smith referring to my article "The Significance of Failing Vision," objects to the use of eserine in iritis with raised tension.

I would like to point out that the article was a somewhat condensed report of a lecture-demonstration to general practitioners mainly from country districts. The advice given to them was to stop the use of atropine and either use eserine or tap the anterior or posterior chamber. These alternatives are temporary measures and when possible the patients should receive specialist treatment. In certain of these cases I have found eserine of definite value. It lowered the tension and enabled the use of atropine to be resumed. Repeated paracentesis may or may not be necessary.

I find that de Schweinitz, Fuchs and Swanzy recommend the use of miotics.

Iritis with high tension usually occurs in chronic iridocyclitis, "serous iritis." In these cases posterior synechiæ are not a marked feature and their development can be watched.

One should not overlook the peripheral synechiæ which may form and may unite the iris to the periphery of the cornea. They block the exit of the albuminous aqueous through the angle of the anterior chamber. Eserine draws the inflamed iris from this angle.

Sometimes it is difficult to decide whether a case is iritis or glaucoma as raised tension may occur in iritis. When other guides fail, it is wise to instil 2% homatropine as a test. One then carefully watches the result. In iritis one will soon see synechiæ appearing as the dilating pupil becomes eccentric. The discomfort of the eye will lessen and the tension remain unchanged. In glaucoma the pupil will dilate more rapidly and will be circular or oval and the tension will rise.

That the treatment of such cases is difficult is undoubted. The most careful observation and the meeting of indications as they arise are necessary.

Yours, etc.,

J. RINGLAND ANDERSON.

108, Collins Street, Melbourne.

August 22, 1926.

QUACKERY.

SIR: Dr. Vallack's letter of June 30, 1926, is timely and unexaggerated. Cannot something be done to protect us from this never ending flood of commercial "literature"?

The amazing thing to me is the number of comparatively intelligent practitioners that absorb this rubbish and straightway proceed to prescribe these productions. Only recently a provincial chemist showed me several prescriptions for "Adreno spermin" that had been ordered by men of repute. He merely smiled tiredly and asked me how long it would last.

Imagine an intelligent individual ordering such hopelessly useless rubbish and yet these same persons would wax hot with righteous wrath in their condemnation against the vogue of the quack and remembering the days of achemy, sigh with relief that he lived in these days of enlightened science.

It might be less comforting to them to remember that their methods were forestalled some time ago. Macbeth's witches prescribed

> "Eye of newt and toe of frog,
> Wool of bat and tongue of dog,
> Adder's fork and blind worm's sting,
> Lizard's leg and howlet's wing"

et cetera. This prostitution of organo-therapy within our midst is infinitely worse than outside quackery, because it is carried on under the ægis of scientific medicine. We are trusted by the laity to do our best for them. Any honest practitioner must recognize his impotence in the face when he is called on to treat numerous maladies, but this fact is no reason that he must lower the insignia of sanity from his mental masthead.

Yours, etc.,

J. NOËL BROWN, M.B., Ch.B.

Melbourne,

August 24, 1926.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the Australasian Medical Congress (British Medical Association), Second Session, Dunedin, 1927, has submitted the following information in regard to the Section of Medicine for publication.

SECTION I.—MEDICINE.

President: Professor A. E. Mills, M.B., Ch.M.

Vice-Presidents: Dr. L. S. Latham, Dr. D. Gifford Croll, C.B.E., Dr. C. T. C. de Crespigny, D.S.O., Dr. W. Horner Nelson, Dr. T. C. Butler and Dr. E W. Gieson.

Honorary Secretary: Dr. C. S. Murray, Forbury Road, Dunedin.

Programme.

Friday, February 4, 1927.

Morning.—Combined meeting with the Sections of Surgery, Pathology and Bacteriology, Preventive Medicine and Radiology. "Goitre: Its Medical Treatment," by Dr. R. R. Stawell.

Afternoon.—"The Metamorphosis of Simple into Toxic Goitre," to be opened by Dr. S. A. Sewell, followed by Dr. F. S. Hansman, Dr. F. Fitchett and Dr. W. Summons, O.B.E.

Monday, February 7, 1927.

Morning.—"Diet," to be opened by Professor A. E. Mills, followed by Professor H. P. Pickerill, Dr. F. L. Apperly and Dr. S. A. Moore. Dr. Apperly will probably be represented by Professor Peter MacCallum.

Afternoon.—"Nephritis," Dr. J. V. J. Duhig, Dr. S. A. Smith and Dr. W. Ray.

Tuesday, February 8, 1927.

Morning.—"Parasites and Blood Diseases."
"The Medical Aspects of Hydatid Disease," Dr. R. R. Stawell, Dr. Neil Guthrie.
"The Value of the Examination of the Cerebro-Spinal Fluid and of the Blood in Treatment," Dr. J. V. J. Duhig.

Afternoon.—"The Diagnosis and Treatment of Pernicious Anæmia."
"Blood Formation," Professor Witherington Stump.
"Blood Transfusion," Dr. A. W. Holmes à Court.

Wednesday, February 9, 1927.

Morning.—Combined meeting with the Sections of Neurology, Surgery and Orthopædics.
"Spastic Paralysis: Medical Aspects," Dr. W. Ray.
General meeting of all Sections: "New Work on Cancer."

Afternoon.—Combined meeting with the Section of Neurology.
"Sequelæ of Lethargic Encephalitis," Dr. H. F. Maudsley, Dr. Marshall Macdonald.
"Localization of Spinal Tumours," Dr. J. F. Mackeddie.

THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

We have been requested to announce that the following medical practitioners have become members of the newly formed Medical Benevolent Association in New South Wales (see THE MEDICAL JOURNAL OF AUSTRALIA, April 24, 1926, page 473). Those who pay ten guineas, are entitled to become life members; the annual subscription is one guinea and members who have paid fifteen guineas in the aggregate, become life members.

LIFE MEMBERS.

Dr. D. Adcock.
Dr. F. Barrington.
Dr. W. J. Binns.
Dr. F. N. Blackwood.
Dr. W. S. Brooks.
Dr. W. F. Burkitt.
Dr. A. W. Campbell.
Dr. W. H. Crago.
Dr. St. J. Dansey.
Dr. J. A. Dick.
Dr. A. J. Dunn.
Dr. R. L. Faithfull.
Dr. J. Foreman.
Dr. F. H. Furnival.
Dr. J. M. Gill.
Dr. Sinclair Gillies.
Dr. J. C. Halliday.
Sir Jarvie Hood.
Dr. J. L. Isbister.
Dr. E. S. Littlejohn.
Sir Alexander MacCormack.
Dr. T. P. McKell.
Dr. J. McKelvey.
Dr. W. McMurary.
Dr. G. Menzies.
Dr. C. Nigel Smith.
Dr. R. B. Wade.
Dr. R. Worrall.

ORDINARY MEMBERS.

Dr. J. G. M. Beale.
Dr. George Bell.
Dr. F. A. Bennet.
Dr. A. J. Bracken.
Dr. E. V. Bradfield.
Dr. P. L. Broadbent.
Dr. K. M. S. Brown.
Dr. G. A. Buchanan.
Dr. H. Bullock.
Dr. A. E. Burrell.
Dr. A. Chenery.
Dr. H. R. Cope.
Dr. W. H. Coutie.
Dr. L. Cowlishaw.
Dr. A. M. Davidson.
Dr. R. L. Davies.
Dr. A. L. Dawson.
Dr. A. W. D'Ombrain.
Dr. Q. Ercole.
Dr. T. H. Fiaschi.
Dr. A. E. Finck.
Dr. W. M. A. Fletcher.
Dr. J. E. Foley.
Dr. R. P. W. Francis.
Dr. W. B. Grant.
Dr. R. A. R. Green.
Dr. J. P. Hocken.
Dr. E. M. Humphery.
Dr. L. P. Johnston.
Dr. E. J. Jenkins.
Dr. W. E. Kay.
Dr. W. L. Kirkwood.
Dr. A. H. Macintosh.
Dr. John MacPherson.
Dr. R. W. H. Maffey.
Dr. F. A. Maguire.
Dr. H. H. Marshall.
Dr. T. M. Martin.
Dr. C. G. McDonald.
Dr. R. J. Millard.
Dr. A. A. Palmer.
Dr. H. R. G. Poate.
Dr. R. A. Robertson.
Dr. C. H. Shearman.
Dr. C. W. Sinclair.
Dr. R. Scot Skirving.
Dr. E. C. Temple Smith.
Dr. H. S. Stacy.
Dr. E. H. M. Stephen.
Dr. H. Thomas.
Dr. R. H. Todd.
Dr. R. C. Winn.

The South Sydney Medical Association has paid a subscription of ten pounds and the Eastern District Medical Association has paid a subscription of five guineas.

A number of medical practitioners have been appointed local secretaries of the Association for the sixteen districts in New South Wales.

Full particulars concerning membership can be obtained from the Honorary Secretary, The Medical Benevolent Association of New South Wales, 30 to 34, Elizabeth Street, Sydney.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on August 9, 1926. The following degrees were conferred:

M.D.: E. S. Morris, J. C. Windeyer.
D.Sc.: P. D. F. Murray.
Ch.M.: F. C. Golding.

A collection of about a thousand books for the Fisher Library was received from His Honour Judge Armstrong.

It was decided to hold the Ceremony of Commemoration of Benefactors in the Great Hall, on Tuesday, October 5, at which the Governor-General will be present. A conversazione will be held at the same time at which graduates of the period 1921 to 1926 will be guests of the Senate and Staff.

Proposals from the Extension Board in regard to visits of overseas lecturers to Australia were approved, and invitations will accordingly be given by the Board to Sir Gilbert Murray, Professor Fay, Professor Bergson and Sir Arthur Quiller-Couch to deliver lectures in Australia.

The following examiners were appointed for the conduct of the forthcoming Final Degree Examination in Medicine:

Medicine: Acting-Professor S. A. Smith, Dr. Cecil Purser, Dr. H. J. Ritchie.
Surgery: Professor F. P. Sandes, Dr. R. B. Wade, Dr. H. S. Stacy.
Obstetrics: Professor J. C. Windeyer, Dr. S. H. MacCulloch.
Gynæcology: Dr. R. L. Davies, Dr. G. Armstrong.
Clinical Medicine: Dr. Sinclair Gillies, Dr. A. W. Holmes à Court, Dr. Hamilton Marshall, Dr. C. G. McDonald.
Clinical Surgery: Dr. G. H. Abbott, Dr. St. J. Dansey, Dr. C. E. Corlette, Dr. A. Aspinall.

AUSTRALIAN ARMY MEDICAL CORPS LECTURES.

AN announcement appeared in our issue of September 11 advertising a series of lectures to be delivered to medical officers of the Australian Army Medical Corps at the Gar-

rison Hospital, Victoria Barracks, Sydney, in September, October and November of this year. The lectures have been arranged by the Deputy-Director of Medical Services of the Second Military District. All officers of the Australian Army Medical Corps are invited to attend. Lieutenant-Colonel H. Poate will deal with war surgery on September 21; Major H. Sutton, O.B.E., will lecture on field sanitation on September 28; Lieutenant-Colonel P. Fiaschi, O.B.E., will speak of prophylaxis and treatment of venereal diseases in the field on October 12; Major-General G. W. Barber, C.B., C.M.G., D.S.O., V.D., Director-General of Medical Services, will take as his subject Australian Army Medical Corps training and mobilization on October 26; Lieutenant-Colonel H. G. Stacy will describe the working of a casualty clearing station on November 11, 1926.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of *The Medical Act* of 1925, as duly qualified medical practitioners:

Caldera, Ciro, M.D., 1910 (Univ. Turin), Brisbane.
Forbes, Brucklay Robertson Vincent, M.B., Ch.M., 1926 (Univ. Sydney), Brisbane.
Kelly, Gerald George, M.B., Ch.M., 1924 (Univ. Sydney), Brisbane.
Moon, Arthur Alexander, M.B., Ch.M., 1926 (Univ. Sydney), Townsville.

Obituary.

WILLIAM ARTHUR HANDCOCK BURKITT.

WE regret to announce the death of Dr. William Arthur Handcock Burkitt, late of Goulburn, which occurred at London on September 7, 1926.

CECIL JOHN DAVENPORT.

WE have to announce with regret the death of Dr. Cecil John Davenport, formerly of South Australia, which occurred recently at Shanghai, China.

JAMES MILLAR MACKAY MUIR.

WE regret to announce the death of Dr. James Millar Mackay Muir which occurred at Wynyard, Tasmania, on August 28, 1926.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants *locum tenentes* sought, etc., see "Advertiser," page xx.

BURREN JUNCTION MEDICAL FUND: Medical Officer.
ISISFORD DISTRICT HOSPITAL, QUEENSLAND: Medical Officer.
QUEEN VICTORIA HOSPITAL, LAUNCESTON, TASMANIA: Resident Lady Superintendent.
ROYAL AUSTRALIAN AIR FORCE: Medical Officer.
ROYAL PRINCE ALFRED HOSPITAL: Two Honorary Assistant Surgeons, Honorary Assistant Radiographer, Honorary Assistant Physician to the Psychiatry Clinic.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE: Three Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

SEPT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Illawarra Suburbs Medical Association, New South Wales.
SEPT. 30.—New South Wales Branch, B.M.A.: Branch: Election of two members of Federal Committee.
SEPT. 30.—South Australian Branch, B.M.A.: Branch.
OCT. 1.—Queensland Branch, B.M.A.: Branch.
OCT. 1.—New South Wales Branch, B.M.A.: Delegates of Local Associations meet Council (First Day).
OCT. 2.—New South Wales Branch, B.M.A.: Delegates of Local Associations meet Council (Second Day).
OCT. 5.—Tasmanian Branch, B.M.A.: Council.
OCT. 5.—New South Wales Branch, B.M.A.: Council (Quarterly).
OCT. 6.—Victorian Branch, B.M.A.: Branch.
OCT. 6.—Western Australian Branch, B.M.A.: Council.
OCT. 7.—South Australian Branch, B.M.A.: Council.
OCT. 7.—Section of Orthopædics, New South Wales Branch, B.M.A.
OCT. 8.—Queensland Branch, B.M.A.: Council.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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